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CONTENTS.

ORIGINAL ARTICLES:—

CRITICAL DIGESTS AND NOTICES OF BOOKS:—

CLINICAL CASES:—

ABSTRACTS OF BRITISH AND FOREIGN JOURNALS:—

LIST OF CONTRIBUTORS.

ALTHAUS, JULIUS, M.D.
BASTIAN, R. CHARLTON, M.D.
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FOX, ARTHUR W., M.D.
FRASER, DONALD, M.D.
GALTON, J. C., M.D.
GOWERS, W. R., M.D.
HORSLEY, V.
HUGHLINGS-JACKSON, J., M.D.
IRELAND, W. W., M.D.
JAMIESON, JAMES, M.D.
JONES, M. HANDFIELD, L.R.C.P.
LAWSON, ROBERT, M.B.
LEWIS, BEVAN, L.R.C.P.
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SAUNDBY, ROBERT, M.D.
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TEALE, T. PRIDGIN, M.A.
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THOMPSON, SILVANUS P., D.Sc.
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URQUHART, A. R., M.D.
WALLER, A., M.D.
WARNER, FRANCIS, M.D.

LIST OF ILLUSTRATIONS.

BRAIN.

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Original Articles.

A PLEA FOR THE NEUROTIC THEORY OF GOUT.

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"The difficulties and refinements relating to the disease itself . . . . I will leave for Time, the guide to truth, to clear up and explain."
A Treatise on Gout and Dropsy, Sydenham, 1683.

I VENTURE in the present essay to put forward some arguments which tend, in my opinion, to sustain the thesis, that gout is a malady of neurotic origin.

In the first place, I wish to clear myself from a charge that may be brought against any one who seeks to promulgate such a view. I must confess that I am not bound to any one-sided theory of disease, in general; that I am not a rampant neuropathologist; and that I am constantly endeavouring to seek exactness and truth in medicine. And, having said this much, I may pass on to state that, having been a pupil and intimate friend, for many years, of the late Professor Laycock, I have not failed to imbibe, and to weigh very carefully, the doctrines laid down by him on many matters of profound interest in practical medicine and pathology. I have carried these views along with me, and have applied them, day by day, to the facts which have come before me in the course of hospital experience. At my hands, therefore, these views have received the roughest and most work-a-day application.

"Gout is primarily and pre-eminently a neurosis." So wrote Laycock to me a year before his death. His great predecessor in the Edinburgh Chair of Practice of Physic, Cullen, taught the same doctrine.

"Gout is manifestly an affection of the nervous system, in which the primary moving powers of the whole system are lodged. The occasional or existing causes are almost all such as act directly upon the nerves or nervous system, and the greater part of the symptoms of the atonic or retrocedent gout are manifestly affections of the same system. This leads us to seek for an explanation of the whole of the disease in the laws of the nervous system, and particularly in the changes which may happen in the balance of its several parts. Of the several pyrexiæ which are diseases of the sanguiferous system, some are with, and others without a considerable affection of the nervous system: pyrexia and neuroses, therefore, are necessarily and unavoidably mixed more or less with one another. Of those which are mixed, gout is a principal instance, in so far as it is an inflammatory disease; like rheumatism, it is placed among the pyrexiæ, but it is among the limits between pyrexia and neuroses, and shows more than any other pyrexia does of an affection of the nervous system."[1]

Cullen states that he adopted his views as to the nervous causes of gout from Stahl.[2]

Within the last few years, large progress has been made in the study of nervous disease, and quite recently, in particular, has light been shed upon various affections of the joints, which can now with certainty be relegated to disorders of the central nervous system.

Few writers have approached this subject since Professor Laycock put forward his views, and these, indeed, were never fully developed by him.

His most minute description is given as follows: "Excessive activity of the nervous system, or of portions of it, becomes a highly predisposing cause of the neuroses, as of

[1] 'First Lines of the Practice of Physic,' vol. ii. part i. chap. xiv. Edited by John Thomson, M.D. Edin., 1827.

[2] 'Theoria Medica Vera,' &c. G. E. Stahl. (Halle, 1737.) "De Doloribus Spasticis Arthritico-Podagricis," § xxxviii. p. 1040.

other general diseases. . . . These excesses, when habitual or long continued, are apt to develop hereditary tendencies. Thus, great mental labour, drunkenness, strain on special nerves, and the like, of parents, are often manifested in children as neuroses. Here the influence of the nerve-centres on the nutrient forces of tissue is shewn, as in hereditary insanity, epilepsy, hysteria, angina pectoris, *and even gouty diseases in general*, which, primarily, are neurose degenerations in the nutrition and transformation of certain tissues." [1]

The exceptions I have alluded to, I must pause to refer to briefly. The first consists in a paper which was written by Dr. Austin Meldon of Dublin,[2] in which he makes objection to the uric acid theory as explaining fully the cause of gout. He quotes cases to show, as is well-known and recognised, that uric acid and urates may exist in the blood to large extent without giving rise to gout, or being the result of gouty taint or inheritance, and he invokes the nervous portion of Cullen's theory to "complete the chain." His theory is as follows. He believes that the presence in the blood of uric acid and of soda, in some form, constitutes the predisposing cause of gout. "Nerve-force, when in a healthy condition, preserves these two in a fluid state, separately, in a condition in which they may be eliminated by the skin, kidneys, or bowels. As soon, however, as this nerve-influence is lessened, these two substances unite in the tissues most removed from the brain and centre of circulation. Irritation and inflammation excite the nervous system to increased energy, and the disease, for the time, is arrested." Dr. Meldon lays stress upon the fact of the attacks occurring mostly at night, when the nerve-force and circulation are feeblest, upon the common implication of the big toe, and notes the marked effect of depression of the nervous power as an important factor in their production. This theory, then, is a compound or humoro-neurotic one.

The second exception I have referred to above is a very notable one. I allude to the views of Dr. Edward Liveing which were published in his masterly exposition of the subject

[1] 'The Principles and Methods of Medical Observation and Research.' Edit. 2 (Edin. 1864), p. 338.

[2] 'Lancet,' vol. ii. (1872), p. 115.

of megrim. Discussing the alliances, and the various symptomatic forms, of megrim, he observes that—"There is much in the history of gout—its hereditary character, limitation to particular ages and sexes, periodicity, explosive character, sudden translations and remarkable metamorphic relations with nervous disorders—*which seems to stamp the malady as a pure neurosis;*" and even the fit itself, with its sudden nocturnal invasion, the late Dr. Todd was accustomed to compare to one of epilepsy or of asthma. Moreover, although the presence of uric acid in the blood of gouty subjects is no longer inferential and admits of ready demonstration, the dependence of the remaining phenomena of gout upon this associated condition, is, to say the least, far from proved; and it is further certain that uric acid is also present in excess under other pathological conditions which have no connection whatever with gout. On the whole, there is much to be said in support of the view that *gout in its various forms is the manifestation of a disorder which has its primary seat in the nervous system itself;* and there is no more difficulty in conceiving that inflammation and pain may be an effect of deranged innervation in the case of arthritis, than in the analogous case of herpes zoster; or that an excess of uric acid should be generated or retained in the system under a similar influence, than that sugar should in the parallel case of the diabetes which follows a lesion in the floor of the fourth ventricle." [1]

As bearing on this subject, Sir James Paget [2] remarks that "disturbance of the nervous system in some form and part may be regarded as a factor in every case of gout. There are reasons enough for thinking that changes in the nervous centres determine the locality of each gouty process, while changes in the relations of the blood and tissues determine its method and effects; and that thus we may explain the symmetries of disease in gout, sometimes bilateral, sometimes antero-posterior, and thus its metastases. But these changes are a part of the pathology of gout which is not yet clinical." [3]

[1] 'On Megrim, Sick-Headache, and some Allied Disorders.' By Edward Liveing, M.D. Cantab. (Lond. 1873), p. 404.

[2] 'Clinical Lectures and Essays.' Edit. 2 (1879), p. 382.

[3] 'Studies of Some Irregular Manifestations of Gout' (1879), p. 93.

In a paper published last year in the 15th Volume of St. Bartholomew's Hospital Reports,[1] I alluded to these views of Cullen, Laycock, and Liveing, and quoted the teaching of Laycock on this subject, showing how he regarded the perverted chemistry of gout merely as an epiphenomenon of a more profound and overruling neurosis; and I ventured to predict that at no distant period Cullen's assertions would be more completely verified and vindicated. Cullen's views have been vigorously combated by Dr. Garrod in his classical work on gout, but I venture to think that with additional evidence now forthcoming, and by the light of other modern doctrines established since Cullen's day, the teaching of that illustrious man, with certain modifications, may not unfairly be sustained at the present time.

Gout has certainly held its place for a long time in humoral pathology, and although Cullen's views tended to overthrow the theory of any special morbid state of the fluids, it does not appear that any marked attention was paid to the nervous part of his ætiology. More stress was laid by his followers upon that portion of his theory which embraced the tone or atony of the system. Cullen held that loss of tone occurred in the extremities, and that this atonic state was communicated to the whole system, but especially to the stomach. He believed that nature restored the lost tone by setting up an inflammatory affection in the extremities.

He further developed from this more fanciful theories, to explain different varieties of gout, such as atonic, misplaced, and retrocedent. Such views as these are hardly perused with patience in these days, and the promulgator of them is naturally regarded as an effete authority.[2] But it is possible, I believe, to rescue from oblivion, and to develop more fully, Cullen's theory as to gout being first, "a disease of the whole system, depending upon a certain general conformation and

[1] Mr. Spencer Wells has called attention to the highly developed nervous system of the gouty, and to the influence of irregular exercise of it, in inducing paroxysms.—'Practical Observations on Gout,' etc. (1854), p. 21.

[2] "The view of the solidists, represented by Cullen, who considered gout to be an affection of the nervous system, has never been able to hold its ground against the various humoralistic theories." Senator, 'Ziemssen's Cyclopædia,' Art. Gout, p. 101, Eng. Translation.

state of body;"[1] and secondly, that it "is manifestly an affection of the nervous system."

The researches of Dr. Garrod seemed to place the modern pathology of gout upon a fresh and secure humoral basis. His facts respecting the uric-acid-excreting function of the kidneys, and the relations between this acid and the disease, have not been controverted, and they constitute a solid advance in medical science, which must ever remain most honourably associated with his name. Still, Garrod, with a degree of caution becoming his scientific position, allows that his views are "in themselves insufficient to explain all the phenomena of gout." He has endeavoured to meet every difficulty which opposed itself to his own theory, and, it must be allowed, with great power and ingenuity. But he has nowhere availed himself of any view which would admit the interposition of nervous influence, and he does not attempt to combat Cullen's particular theory of the specific involvement of the nervous centres. Sir Charles Scudamore, who criticised Cullen's views, also took no heed of the alleged implication of the nervous system, and adopted a humoral theory, starting from the stomach.[2] In more recent times too, the late Dr. Murchison gave in his full adherence to a humoral theory of gout, and expressed himself thus: "I hold that what is called a 'gouty diathesis' always indicates, and is the result of hepatic derangement, and that many symptoms commonly referred to gout, would be more correctly ascribed to disorder of the liver. . . . Gout itself is merely one of the results of lithæmia."[3] The idea that gout was in any way a manifestation of disturbed nervous relations, seems never to have been contemplated by that very thoughtful and philosophical physician.

In a suggestive communication by Dr. Ord,[4] on the relations of uric acid to gout, he offers the view that the malady is a

[1] Op. cit., par. DXXX.

[2] 'A Treatise on the Nature and Cure of Gout and Gravel,' edit. 4 (1823), pp. 10 and 147. Cullen's "third observation" was, that the stomach was the internal part most frequently and considerably affected by gout, yet he repudiated any humoral pathology in connection with it.

[3] 'Lectures on Diseases of the Liver.' Edit. 2 (1877), p. 568.

[4] 'Medical Times and Gazette,' vol. i. (1874), p. 233.

special form of degeneration or want of tissue-organisation in remote, and lowly vascular parts. He believes that the uratic deposits result from either general or local disintegration, and are not to be regarded as significant of their elimination from the blood: further, that the local processes are not dependent on these deposits, and that the latter are not the result of the inflammation. He takes cognizance of nervous influence so far as to affirm that "all authors, in one way or another, admit the direct influence of the nervous system" (which statement I can hardly agree with), and he believes that local gouty "degenerations and inflammation tend to infect the rest of the system through the blood, *and to set up similar actions elsewhere through reflex nervous action.*"

Dr. Ord's theory is therefore mainly humoral, and he is chiefly concerned to combat Garrod's views.

To discuss more in detail the question of the humoral element in the pathology of gout, is not within the scope of the present essay.

It is, without doubt, the case that, hitherto, no theory has been set forth which appears to embrace all the multiform phenomena of gouty disease. The greatest advance of modern research has been to establish the certainty of some special relation to it, in the greater number of instances, of uric acid, and, so far, there is clear warrant for retaining a measure of humoral pathology in our conception of the malady. There is still much to be done in elucidating the causes of the renal inadequacy which must be taken into the consideration of a comprehensive pathology of gout.

The best approach to the line of argument I purpose to take up, will manifestly be to review the special characters of neuroses in general, and then to examine coincidently, how far the well-ascertained features of gout conform to such characters.

Before proceeding to this analysis, I would first assert that gout is something beyond the mere resultant effects of aberrant relations of uric acid; that it consists in something more than a perversion of animal chemistry; that it is not, by any means, to be explained as an outcome of gastric or hepatic distemper; and that it is not the appanage only of the middle-

aged or elderly high-liver and intemperate drinker, because, as is well known, it affects also, sometimes in early life, the high-thinker and the laborious bread-winner. Without doubt, while accepting all (and that is much) that it is good for, one is impelled to look beyond what may be termed the chemical pathogeny of gout. The researches into the nature and functions of the nervous system, as carried out during the past quarter of this century, come to our aid at this stage of our inquiry, and, amongst these, we have learned two main and important points respecting the neuroses in general. The first is, that they may be primary or central, and the other is, that they may be secondary or induced. In other words, it may be averred that a neurosis is implanted, or a tendency to it established, and this shall be handed down, hereditarily passed on, and thus, a diathetic tendency be formed; or, owing to some toxæmic condition or blood-degeneration, a secondary or induced neurosis may be established.

I shall endeavour to sustain the neurotic theory of gout upon this basis.

Representing special conditions, or rather special morbid modes of evolution, of nerve-force, neuroses are implanted in the individual as a part of his intimate nature. They belong to the individual, and are characteristic of him in the same degree that are his features and other physiognomical traits. An implanted neurosis is, as it were, the representative of a morbid physiognomy for the cerebro-spinal axis. A neurosis, then, is a peculiar disposition, or tendency, on the part of the nervous system, or some definite tract of it, towards morbid evolution or manifestation of nerve-functions. It does not necessitate the existence of any coarse disease, directly obvious to the eye, but it is a more or less abiding condition, ready to come into action upon suitable provocation.

It is specially characteristic of neuroses that, being thus primarily impressed upon an individual, they tend to be transmitted by heredity. It has been alleged that the female sex is more neurotically disposed than the male; but facts do not support this opinion thus broadly put forth. Certain neuroses appear to prevail with greater frequency in males, and others in females; and not only so. but in the case of those

that are common to both sexes, the manifestation occurs at different epochs of life.

Thus, some outbreaks of neurotic disorders are seen to occur at the several septennial climacteric periods, at the times of dentition, puberty, and often at the grand climacteric. In this manner, an element of distinct periodicity attaches to neuroses in general.

Further, a most marked feature in all neurotic affections is that of paroxysmal tendency. Thus, there is the abiding element, with proclivity to recurring outbreak.

Again, it is certainly known that a law of alternation, or substitution, prevails in neuroses, and thus we meet with certain affections in the parent or ancestors, and with others, equally neurotic, in the collateral relations or descendants. We thus have to deal with distinct types of nervous impression. These abiding conditions are more or less prone to be excited into activity according to various circumstances.

It is not difficult to understand the course pursued by a neurotic taint once laid down or impressed; but it is not so easy to conceive the original implanting of such a morbid functional tendency. The mischief, however, is constantly originating in individuals, and as constantly undergoing further development, modification, or even repression.

Excessive activity of the nervous system, or of any part of it, as Laycock has shown, becomes a highly-disposing cause of the neuroses. Habitual or prolonged excess comes thus to develop hereditary tendency. Thus, *undue mental labour, gluttony, alcoholic intemperance, debauchery, and other indulged evil propensities in the parent, come to be developed into definite neurotic taint and tendency in the offspring.* Particular examples of this are not far to seek, and amongst them comes out the disorder so widely and variously manifested as gout. According to this view, for which I plead, gout appears as a diathetic neurosis, and a link in the long chain of its phenomena, so long missing, is now forthcoming.

I have already stated that there is clear warrant for retaining, as *part of the pathology* of gout, a humoral hypothesis, and it may perhaps be applied and relegated to its proper place, as follows. Granting that gout in any individual is the outcome

of a central neurotic taint, we have the ordinary manifestations of it more or less severe. This we may term primary or central gout. The tendency may be transmitted or modified, or, conceivably, may be allowed to die out.

In another individual, gout may "grow up" where previously there was no neurotic taint or tendency. A patient is commonly said to earn his gout by high-living and over-indulgence of appetites. In this instance a morbid blood-state is induced; excess of uric acid is generated, and hyperinosis supervenes.

But is this all? Is this enough to generate all the phenomena we recognise clinically in gouty disease? I believe not. We are compelled at this point to widen our view, and are driven, perforce, to invoke the operations of the nervous system. Having arrived thus far at nothing beyond a special toxæmia, we must drop humoral pathology, and seek for the effects of the blood-dyscrasia upon the nerve-centres. And we have full warrant for this course in contemplating the analogy of other toxæmic states, together with their effects upon the nervous system. The nutrition of this system is plainly affected by morbid blood-conditions, and, thus, expression is given to such poisoning in the form of convulsion and other nervous symptoms.

I believe, then, it may be conceded that a secondary affection of some nerve-centre occurs as a consequence of the altered blood-state *ab intra*, and that thus the order and particular process of the gouty attack is evolved. This we may term secondary or acquired gout. A diathetic neurosis is thus impressed upon the individual.[1]

It is certainly a matter of much interest to study side by side with gouty processes the several joint-affections, or arthropathies, which have come to be regarded of late as of distinctly spinal or otherwise nervous origin. It seems impossible to separate gouty arthritis from this connection. And if it be conceded that this particular form, which is but one

[1] The frequency and severity of gout in England is explicable on the view of the impressed neurosis. The habits leading to gout,—high-living, intemperance in strong drinks (malt-liquors and wines), along with much mental energy, have certainly prevailed more, and amongst larger classes, in England than in either Scotland or Ireland.

of many others, is truly and directly dependent upon nerve-influence, the greatest part of the difficulty in establishing a neurotic theory of gout is forthwith removed. I suppose no greater obstacle has stood in the way of the acceptance more generally of a nervous theory of this malady, than the impossibility hitherto of connecting arthritic disposition with any form of neurosis. So many of the other, and less obvious, manifestations of gout are plainly dependent on nervous influence, that the whole phenomena now appear to fall more naturally into their places.

It is, however, only right to mention here that thoughtful physicians have long ere now conceived the special action of nerve-influence on joints, and of arthritic affection on nerve-centre.[1] The relation sometimes existing between rheumatic fever and chorea is an example in point, as Dr. Liveing has shown.

Much light has been thrown of late upon spinal arthropathies by the researches of Charcot, Ball, Weir Mitchell, and of Dr. Ord. The latter has recently contended for a more scientific revision of our present views upon the pathology of osteo-arthritis or rheumatic gout.

Without elaborate reasoning, Dr. Noël Gueneau de Mussy, of Paris, expressed, some years ago, his opinion that the latter affection was related to gout, as a sort of cousin, but without in any way holding the view that the disorder was a compound of rheumatism and gout.[2]

Dr. Ord's views are not only eminently ingenious, but they accord remarkably with well-observed clinical facts, not hitherto correlated.

As Sir James Paget has remarked, the changes in the nerve-centres which determine the locality of the gouty process, are a part of the pathology of gout which is not yet clinical. They are, therefore, no more than speculative at present, but we gain much from the prosecution of an inquiry in this direction.

With respect to the particular locality affected in the

[1] Liveing, op. cit. p. 247. Weir Mitchell, Charcot, &c.

[2] Mr. Hutchinson believes in a "basic diathesis," for both gout and osteo-arthritis.

arthropathy of locomotor ataxia there is some discrepancy of opinion. Charcot has declared for implication of the anterior cornua of the spinal chord. Dr. Buzzard, however, has not confirmed this opinion, and, guided by the noteworthy frequent association of gastric crises with these joint-affections in this malady, as previously observed by Dr. Ball,[1] has suggested a sclerosing lesion, involving the roots of the vagus in the medulla oblongata, in close relation to some trophic centre that may be localised there, presiding over the osseous and articular systems. And he further indicates the bond that may thus exist between implication of joints and such metastasis as may occur to the heart in rheumatic fever: also the occurrence of hyperpyrexia, which is sometimes present in such cases. We have yet to seek for this hypothetical nutrient centre for joints, but in the meantime we are fairly warranted in widening our view, and in directing attention to the high significance of predicating such a trophic centre.

"Discovery by true analogies is always progressive, . . . one analogy leads on to another investigation and arrangement of phenomena, and another analogy."[2]

It remains now to be shown, more in detail, how the phenomena of gout conform to the recognised manifestations of the neuroses in general.

It can be shown, I believe, that the plea for the neurotic element in true gout is not difficult to establish.

First, there is to be considered the marked tendency of gout to be hereditarily transmitted. This is notorious. The disorder may pass from either parent, and may be mingled with other taints and tendencies passed on from the progenitors. The outbreak may occur in slight or in graver degrees, and may be deferred, overtly, till even the thirteenth climacteric period. Thus, the first plain attacks of gout may not appear till the patient is sixty or over ninety years of age. In all such cases, however, I am convinced that many minor tokens of the disorder have been overlooked in previous years, all of which are sufficiently obvious to the trained clinical eye.

[1] 'Med. Times and Gazette,' vol. ii. (1868); vol. ii. (1869), p. 498.

[2] Laycock, op. cit. 190.

As a rule, the manifestations are prone to occur at definite ages in each sex, most commonly in the fourth decade in men, and in the fifth in women. My own experience seems to show that gout is increasingly frequent in men early in the third decade.

Certain peculiarities attending gouty transmission are deserving of study. Mr. Hutchinson has called attention to one of these in a suggestive lecture published four years ago.[1] He expresses his belief that what is transmitted is not the active gouty dyscrasia itself, but rather a susceptibility to the influence of certain exciting causes, together with some peculiarly disordered condition of the assimilating and excretory viscera, which renders them unable to deal with particular articles of food. Now, this special susceptibility to definite exciting factors is neither more nor less than a nervous peculiarity, of which the chief character is its liability to break away in certain morbid directions,—its *instability*, in short. This is, I submit, the gouty neurosis. Mr. Hutchinson further believes that gout is wont to show itself with greater frequency and in more marked form in the younger than in the older members of a gouty family, the diathesis strengthening in the parent with advancing years. I think I can confirm this observation.[2] Resemblance to the gouty parent has been specially recognised in those of the offspring most distinctly affected.[3] In other members of the family the tokens of gout may be present, but less marked. These facts are, of course, in accordance with ordinary laws of hereditary transmission. Dr. Wickham Legg has called attention to the fact that gout, like hæmophilia, pseudo-hypertrophic paralysis of Duchenne, and some other affections, is not unfrequently found to be transmitted by the female line, although especially manifested in males, the mothers themselves being unaffected.

A noteworthy feature in gouty ailments is their sudden supervention. As in epilepsy, not uncommonly, the patient often feels remarkably well, and realises his sense of *bien être*, before the outbreak suddenly takes place. This euphoria, or

[1] 'Medical Times and Gazette,' vol. i. p 543 (1876).

[2] Cases illustrating this are given by Mr. Spencer Wells, op. cit. p. 18.

[3] 'Medical Notes and Reflections on Hereditary Disease.' Sir Henry Holland, Bt., M.D., F.R.S. Edit. 3 (1855), p. 29.

delusive corporeal satisfaction, is itself a nervous derangement. Explosiveness is a distinct feature in several of the neuroses, and attaches to such ailments as angina pectoris, asthma, epilepsy, and various neuralgiæ.

The time of the occurrence of the attack is also strongly marked. The majority of the outbreaks take place in the early morning. This is true both of grave and classical cases, and also of many of the minor forms of gouty trouble. The same thing is met with in asthma, neuralgia, and in epilepsy. The pyrexia proper to acute gout is paroxysmal, with remissions, and the pain of gout is likewise paroxysmal. One is here reminded of the influence of marsh-poison upon the nervous centres.

This paroxysmal, no less than the periodic, element in gout, stamps a nervous character upon the malady, and binds it in alliance with other neuroses.[1]

An important connection of the same kind is seen in the unquestionable commingling of gout with other well-recognised neuroses. Thus, hemicrania is sometimes distinctly a manifestation of gout in both sexes, and may be the form of neurosis impressed upon an individual whose parent was gouty, or may itself alternate with gouty arthritic attacks in the same person.[2]

It is not far to seek for an allied condition of trophical lesion in herpes zoster, itself the outcome of disordered innervation.

The doctrine of metastasis must next be considered in relation to gout. The humoralist seeks to explain this clinical fact upon his theory, but such is manifestly insufficient to account for the phenomena. It must be conceded that some nervous law regulates the occurrence of shifting inflammation. It has been supposed to be due to reflex influence. Some distinct predisposition to take on the morbid action exists in the part selected apparently by caprice. The same class of tissue is apt to suffer. Thus, the gouty or rheumatic process flies from joint to joint, or, as in gouty phlebitis, from vein to

[1] Vide Scudamore, op. cit. p. 152.

[2] Stahl, op. cit. § xxxvi. Trousseau, 'Clin. Méd.' Liveing, op. cit., &c. Sir H. Holland, op. cit., 'Relation of Asthma to Gout,' p. 36.

vein, sometimes symmetrically, but not always. The serous and fibro-serous structures suffer especially, but also mucous surfaces. Laycock has shown how these several tissues are related embryologically, and are thus prone to suffer in common when diathetically impressed.[1]

Localised trophical changes follow locally acting causes of depressed nervous power. Thus, impairment of certain centres may lead to the specific nutritional changes witnessed in metastases, and thus the apparent capriciousness is explained in this process.

Amongst the nervous symptoms of gout must next be considered the occurrence of certain sensory perversions, such as tingling and numbness of the fingers and toes, sensations of heat in the palms, thighs, and soles (paræsthesia), and tickling in the throat. As pointed out by Sir James Paget, "gout affects the sensory much more than the motor elements of the nervous system," and he remarks, too, that the pain of acute gout is seemingly out of all proportion to the amount of inflammatory process in the affected part. So, too, all other disorders, modified by gout, seem to be especially painful, for example cancer, as pointed out also by Paget.

Grinding of the teeth is met with in the gouty. Graves first observed this.[2] Garrod has not met with it. Dr. Donkin has lately recorded cases associated with somnambulism,[3] and I have intimate knowledge of two others in which the same phenomena are manifested—the grinding of teeth and somnambulism in a sister, and the talking in sleep in the brother. The maternal grandfather and the mother are distinctly gouty. Cramps in the muscles of the legs, and priapism, are amongst nocturnal manifestations in the gouty. Of insomnia, due to gout, there is much to say. It was originally noted by Cullen, and it conforms remarkably with other periodic neurosal phenomena.[4]

Gouty neuralgia is largely recognised, and is known to be both severe and prone to recur. It is frequently occipital,

[1] Op. cit., p. 196.

[2] 'Clin. Med.,' p. 351 (1864 edit.).

[3] 'Brit. Med. Journal,' Feb. 21, 1880, p. 279.

[4] Vide Author's paper, 'St. Barth. Hosp. Reports,' jam cit., p. 105.

and is met with in the heel, tongue, breast, aud more often in the great sciatic nerve. One proof, amongst others, of the truly gouty nature of these is gained from the fact that they yield most readily to anti-gouty medication, and another lies in the frequency with which they are provoked by conditions which elicit other gouty processes.

Amongst the strongest evidences of gout depending upon nervous influences, are the unquestionable facts bearing upon the induction of its attacks.

The influence of many of the existing causes of gouty paroxysms illustrates well the explosive character of the malady. As Sydenham expresses it, before the onset of an attack, "totum corpus est podagra." The precipitation of the seizure sometimes ensues almost immediately upon the provoking cause. In a large number of instances, the latter is of a nature *to depress nervous power.* Thus, unwonted muscular energy, prolonged exercise, stirring emotions, fright, undue excitement, venereal excess, rage, worry, and vexation, are all excitors of gouty paroxysm. So, too, sudden shock to the body, as from injuries and surgical operations, will evoke gout. Dietetic errors are well recognised as factors; thus, a full meal, and excess, or mixing, of strong liquors, will act in upsetting the equilibrium of a quiescent gouty habit. It will be conceded that many of the causes just enumerated are equally potent to elicit manifestations of other neuroses, such as epilepsy, asthma, hemicrania, and angina pectoris. The provoking agency, however, need not always be primarily exhausting. In proof of this, the outbreaks of gout following hydropathic treatment, internally or externally, may be instanced, and, indeed, the causal element need only be such as shall induce some change in the acquired vital habits.

Thus it is, that the subjects of the neuroses hold much of their comfort in life by following a very equable routine. They are prone to give way under any extraordinary pressure.

These considerations explain, in part, why men are more liable to gout than women. They carry on the world's rough work; are engaged in more exciting occupations, and have commonly the greater burden of anxiety to bear.

The more sedentary the occupation, the more profound

the mind-working, and the more intense the strain of life, the greater the tendency to nervous depression, and to the peculiar form of its expression in gout. If to such habits be added high-living, as often occurs in the cases of eminent statesmen, lawyers, and speculators, no link is wanting in the chain of causation, and all the elements for gout are present.

Climatic influence is important amongst these agencies. The dull and "shifty" weather, and the cold east winds of northern latitudes are certainly bad for gout. The nervous depression ensuing upon months of sunless skies—negation of light powerfully lowering nervous tone—is too little regarded as an element of devitalisation in England. The cutaneous eliminant power is checked, and so, aberrant chemical relations are apt to be determined in any parts specially prone to gouty invasion.

The same mal-determination ensues upon the suppression of various discharges, whether from the uterus, from hæmorrhoids, or other sources.

The mental condition of the goutily-disposed is a subject worthy of attention in relation to the pathology of the ailment.

Hypochondriasis has long been associated with gouty taint. It commonly precedes an outbreak, and disappears subsequently. A tendency to sighing has also been observed, and is a plain indication of nervous exhaustion. Hysteria has also been observed to precede gouty attacks in women, and to disappear with the onset of articular symptoms.[1]

Irritability of temper is a proverbial condition in the gouty, and furious outbursts of this kind appear to be, at times, a metamorphic substitution for a more overt and regular attack. It is important to know that many of the minor, but none the less well-marked, phases of gouty paroxysm are in no degree arthropathic. Much error in diagnosis has arisen from taking no heed of any but articular symptoms when searching for gouty tokens in a given case. These less classical attacks very commonly precede the onset of typical ones at a later period in life.

The necessity for prompt recognition of these less well-

[1] 'On the relations between Gout and Hysteria,' *vide* 'Treatise on the Nervous Diseases of Women' (p. 163). By T. Laycock. 1840.

expressed symptoms is obvious, if good treatment is to be applied.

Epilepsy has been known to disappear on the supervention of gout.

Sensations of giddiness and dimness of vision, not uncommon in the gouty, are noteworthy in relation to nervous symptomatology.[1] So, too, the disturbances of the cardiac rhythm, and the co-existent (neurotic) vascular throbbings which are sometimes met with. The cardiac irregularity has been noted to cease with the induction of a regular attack. Dysphagia was noted in connection with gout by no less careful an observer than the late Dr. Brinton.

A consideration of the effects of lead-impregnation in relation to gout, and of the certain liability of the gouty to be more readily than others influenced by lead, leads to the belief that the nervous system is specially implicated in these relations. The fact is, that the blood is imperfectly freed from uric acid in cases of lead-poisoning, and that gout is thus quickly evoked. Dr. Garrod has fully established these facts, and all physicians now recognise them. The lead-influence, clearly through nervous agency, induces the measure of renal inadequacy which is, probably with correctness, acknowledged as a factor in gout. And the knowledge that this metal is capable of setting up special paralysis, epilepsy, coma, and other cerebral phenomena, is of the largest interest in relation to this subject.

I now approach a point in connection with the whole pathology of gout which merits much consideration. The connection of diabetes with gout has been recognised for some years.

I object to the term diabetes as applied to the special form of glycosuria associated with gout. The disorder is met with in certain members of gouty families, some having regular gout, and others manifesting less regular gout, or this alternating with glycosuria. I believe that many cases of temporary glycosuria are due to gouty conditions. The fleeting presence of glucose in the urine of many elderly persons may be thus explained. It has long been recognised that such an affection, which, in many instances, is undeserving of the name

[1] Trousseau, Murchison, Paget, op. cit., H. Mayo, F.R.S., 'Philosophy of Living,' p. 24 (1837).

diabetes mellitus, for the simple reason that there is no *diabetes* in the strict sense of the term, is not really a grave one. *The presence of glucose is found to alternate with that of uric acid.* In the aged but little importance should be attached to the symptom. Charcot testified to this, some years ago, in his excellent lectures on the maladies of old people, his experience being gathered at that fertile school of study—the Salpétrière. In persons under forty years of age, however, glycosuria, even of gouty origin, is a most grave matter, and merits the closest attention, since it may eventuate in confirmed diabetes. It is the rule to find that the quantity of urine passed is not much, if at all, above the normal, but the specific gravity may range from 1·035 to 1·050. An anti-gouty treatment is called for, for the glucose may otherwise only give place to uric acid or increased azoturia, and the gouty habit has rather to be attacked than the glycosuria.

Dr. Lauder Brunton has called attention to this class of cases.[1]

The alliances of gout and diabetes are sufficiently intimate. In both the doctrine of heredity applies, and the nervous system is involved. The same habits lead to each, the same classes of person are affected, and the same exciting causes are potent to evoke both. A consideration of these facts naturally leads to the belief that the portions of the nervous system involved in each cannot be far apart from one another. The medulla oblongata, the sympathetic and splanchnic nerves have been found chiefly affected, and the spinal chord likewise in some instances. The point for the diabetic puncture in the medulla is believed by physiologists to correspond to the vaso-motor centre in the same structure.

Guided by these facts, and by the knowledge that the glycogenic function of the liver is under nervous influence, by the advancing theories which refer special arthropathies likewise to the same influence, and bearing in mind Dr. Buzzard's views, previously stated, in connection with the gastric crises so commonly associated with the arthritis of loco-motor ataxia, I come to the conclusion that the portion of the nervous system which is specially predisposed to the irregular mode of action known as gout, has its seat or centre in the medulla oblongata.

[1] Art. "Diabetes Mellitus," Reynolds' 'Syst. of Med.,' vol. v. p. 381 (1879).

A point of difference between the arthritic affections which are now referred to nervous influence, and those manifested in gout, is found in the fact, that the latter appears to have an elective affinity, often unilateral at first, for the smaller joints, especially that of the big toe, while most of the others influence the larger ones. Herein, perhaps, lies part of the specificity of gout.

The trophical results of the latter are seen impressed upon the physiognomy, and upon certain tissues, in a manner extremely definite and characteristic.

Thus, are found the large head, the thick hair, with tendency to early greyness, the large, full veins, the long uvula, the soft, smooth skin, the thickened extremity of the nose, and the lineated, brittle nails.

Lastly, I may add an argument in favour of the theory, adduced in this essay, from the therapeutical side.

The universally acknowledged specific action of colchicum in gout is known, owing to Garrod's researches, to be due to no power which it possesses of causing elimination of uric acid. Gouty inflammation is therefore influenced by it without reference to the secondary aberrant relations of uric acid. The active principle, or alkaloid, of the drug colchicia, is a member of a nitrogenised group of bodies to which veratria, strychnia, quinia, and morphia have close chemical alliance.[1] They all powerfully affect *the nervous system.* Colchicum acts very promptly, and affords often decided relief to the intolerable pain of the gouty process. When taken in health, in small doses, Dr. Meldon and others have found that it induces a general glow at the surface of the body, diaphoresis, throbbing of the blood-vessels and palpitation. Subsequently, there is reduction in the force and frequency of the pulse. Dr. Meldon observed in his own case an invigoration of his mental energies. In larger doses, the effects are most marked along the whole tract supplied by the vagus, and thus, cardio-vascular, gastric and enteric symptoms ensue.

The peculiar benefit derived from this drug is not secured in any other form of inflammation, and thus it is plainly

[1] Vide 'Lectures on Pathology and Therapeutics.' (London, 1867; p. 137.) H. Bence Jones, M.D., F.R.S.

specific. Its cherished action is doubtless exerted upon the vaso-motor nerves.

The manifestly good influence of all agencies which cheerfully inspire the mental condition in the goutily-disposed, must not be omitted from consideration amongst the *juvantia* both of prevention and cure.

The points in this thesis which I have endeavoured to sustain, I now place categorically under the following heads.

First.—I contend that the diseased conditions which are recognised as of unequivocal gouty nature, are primarily dependent upon a functional disorder of a definite tract of the nervous system, and that, thus, gout is a primary neurosis.

Secondly.—That there is much in the nature of the malady itself, and much evidence forthcoming by way of analogy, to warrant the conjecture that the portion of the nervous system specially involved is situate in some part of the medulla oblongata, where, possibly, may be placed a trophic centre for the joints.

Thirdly.—That the gouty neurosis may, like others, be acquired, intensified, and transmitted, also, that it may be modified variously, and commingled with other neuroses; that it may suffer metamorphic transformations, or be altogether repressed.

Fourthly.—That this diathetic neurosis imposes its type upon the affected individual in definite nutritional modes, affecting the assimilating and excreting powers, exhibiting marked peculiarities in nervous impressibility, and determining, in more or less degree, a physiognomy of the gouty.

Fifthly.—That a large part of the phenomena known as gouty, are due to perverted relations of uric acid and sodium salts in the economy, resulting from the morbid peculiarities mentioned under the last head. Thus, there is excess of urate of soda in the blood before, and during, gouty explosive manifestation, and there is determination (by nervous influence, in all probability) either of this salt to the affected part (Garrod),[1] or there is a too free formation of it at these inflammatory

[1] Op. cit.

points, whence it deposited locally, and also set free into the circulation (Ord).

The renal excretory power for uric acid appears to be temporarily inhibited as part of the process of gouty paroxysm. This measure of renal inadequacy would appear to prevail in varying degree as a part of the specific neurosal disorder. In chronic gout, when structural disease has occurred, either tubal, with deposition of urate of soda, or interstitial, with shrinking of the organs, the renal inadequacy may admit of more mechanical explanation.

Sixthly.—That in Primary, or inherited, Gout, the toxæmia is dependent on the gouty neurosis; is the outcome, in whatever degree, of it, and is therefore a secondary manifestation.

Seventhly.—That, in what I term Secondary, or acquired, Gout, the toxæmia is directly induced by such habits as overload the digestive and excretory organs, and constantly prevent complete secondary disposal of nutritional elements of food; that if, together with such toxæmia, distinctly depressing and exhausting agencies, affecting the nervous system, come into operation, the special neurotic manifestations of the gouty diathesis will occur, and be impressed more or less deeply upon the individual and his offspring.

Eighthly.—That this theory of gout, better than any other, correlates all the known factors concerned in the production of the varied symptoms of the malady; and while it displaces its humoral pathology from the preeminence it has so long occupied, it takes full cognizance of it, and seeks to place it in a clearer relation to the phenomena of the disease.

Ninthly.—That if it be desirable to refer various maladies to their distinct place in pathology, without reference merely to their chemistry, histology, or neurology, the affection known as gout may perhaps most correctly be relegated, along with some others, to a class of diseases which may be termed neurohumoral.

Tenthly.—An argument is adduced from the *juvantia* afforded by colchicum, in favour of the theory which has been set forth.

THE CONDITIONS OF THE UNIPOLAR STIMULATION IN PHYSIOLOGY AND THERAPEUTICS.

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By electrotonus is meant that condition of altered irritability of a nerve which is produced by the flow through it of a current of electricity; and this altered irritability manifests itself by increased or diminished reaction of the nerve to the various stimuli, whether mechanical, physical, or chemical, which may be applied to it.

Du Bois Reymond, who introduced this word in physiology,[1] at first implied by it merely the changes in the electromotive manifestations of the nerve which accompany the changes in its irritability. A clear recognition of the latter, says Hermann,[2] does not occur in any of the older physiologists, nor in Du Bois Reymond's great work. Valentin[3] was the first to show that the polarised portion of the nerve does not readily transmit impulses from above; and that the irritability of the part below it is diminished when the polarising current is centripetal, or ascending. Eckhard[4] observed that when the current is descending, an opposite condition of augmented irritability below the polarised portion existed. Hence he formulated as a general law that irritability is increased beyond the kathode, or negative pole; diminished beyond the anode, or positive pole.

Pflüger, by a series of admirable experiments, eliminated all the sources of error which beset this difficult field of inquiry, and resolved all the phenomena into his dictum:—The irri-

[1] 'Untersuchungen über thierische Elektricität,' 1848, 1849.

[2] 'Handbuch der Physiologie,' vol. ii. part i. p. 41, 1879.

[3] 'Lehrbuch der Physiologie,' 2nd Ed., 1848.

[4] 'Beiträge zur Anatomie und Physiologie,' 1855.

tability is increased on either side of the negative electrode, diminished on either side of the positive. Or, in other words, the region of the kathode is thrown into a state of katelectrotonus, that of the anode of anelectrotonus. In the intra-polar region the two zones meet at a point of indifference, where irritability remains normal. This point is the nearer to the kathode the stronger the current, and *vice versâ.*

Whenever a galvanic current of sufficient strength is made or broken through a portion of a motor-nerve, a contraction in the muscle occurs. It was one of the earliest observations made that a contraction appears earlier (i. e. with a weaker current) when the current is descending (i. e. with the kathode peripherally and the anode centrally placed) than under the opposite conditions (Pfaff, in 1795). Ritter (1798), and Nobili (1829), with a host of other observers, confirmed this fact, and elaborated what are known as "laws of contractions;" that is, tables of the reactions which the exsected nerve yields to make and break off the ascending and descending currents, at different stages of its diminishing vitality. That such researches must have been limited is made at once evident by the consideration that the first constant galvanic element was invented in 1836 by Daniell. Yet on the whole it is possible to reconcile the main results obtained up to the time of Du Bois Reymond,[1] with those of Heidenhain,[2] who substituted "stages of current strength" for the "stages of irritability" of his predecessors. But it would be transgressing our present limits to delay on these topics, and we at once reach Pflüger, to whom we owe also the expression of the phenomena of nerve-reactions to different current-strengths in their simplest formula :—

Current.	Ascending Current.		Descending Current.	
	Make.	Break.	Make.	Break.
Weak	Contraction	Rest	Contraction	Rest
Medium	Contraction	Contraction	Contraction	Contraction
Strong	Rest	Contraction	Contraction	Rest (or weak C.)

[1] Loc. cit. [2] 'Archiv für physiologische Heilkunde,' 1857.

Pflüger did not remain content with leaving his law of contraction and his law of electrotonus side by side; but by a further induction reduced the one to the other, and showed in the happiest manner that a nerve is stimulated by the apparition of katelectrotonus, and the disappearance of anelectrotonus; that is, by the passage of a lesser to a greater degree of irritability. In other words, that the stimulation occurs at one electrode only: at the kathode on closing, at the anode on opening the current. He completely exposed the fallacy of the views of the previous writers, who had all explained by the differences in the direction of the current itself the different results they obtained by the making and breaking of ascending and descending currents; and showed that it was the relative position of the two poles *to one another*, and not to the direction of the nerve-impulse, that was the condition upon which depended the variations in the results obtained.

His argument was based on the facts that the closure of the current is a more powerful stimulant than the opening; that anelectrotonus takes more time and stronger currents to develope than katelectrotonus; and that immediately after the opening of the polarising current the katelectrotonic zone passes into a condition of transitory diminution of irritability, or "negative modification," whilst the anelectrotonic zone passes immediately into one of increased irritability, or "positive modification." Thus it becomes clear why weak currents excite the nerve at the closure only: anelectrotonus is not sufficiently developed by them to cause a contraction on its disappearing, nor to prevent the transmission of the katelectrotonic stimulus from above. Strong currents, on the other hand, develop a powerful anelectrotonus, and negative variation, which arrest the make, and break impulse from above respectively. Medium currents allow a sufficient anelectrotonus to set in which will stimulate on disappearing; but not to arrest the central katelectronic impulse, whilst the negative modification of the katelectrotonic region also is too weak to arrest the stimulation produced by the disappearance of anelectrotonus on the breaking of the descending current.

In presence of the clear fact that, according to Pflüger, stimulation is produced at the kathode on closing, at the anode on

opening of the current, and at the former more readily than at the latter, it is difficult to see how many electrotherapeutical writers have been able to continue speaking of the results obtained by physiologists as dependent on the direction of the current with reference to the nerve; and to place their so-called "polar method" in a position of antagonism to the "physiological method."[1] In the latter, it is true, in order to eliminate many disturbing elements, such as the influence of the central organs, the action of derived currents, &c., the nerve is usually exsected, and both poles applied to it directly; but apart from these differences of manipulation, there is no such fundamental difference between the two methods as has been claimed. Let us first throw a retrospective glance into the origins of that polar method, the subject of so many misunderstandings and passionate recriminations.

Among the earlier observers we come across but few and vague data concerning the effects of the galvanic current on the living body. Ritter[2] noticed that a strong current flowing from hand to hand produced in the arm up which it flows a sensation of increased mobility, and *vice versâ*. Matteucci[3] repeated the observation of Nobili, that tetanised frogs' legs were quieted by an ascending current, and even proposed therapeutical applications of this fact.[4] Valentin[5] is the first to make an explicit statement on the subject. He finds that in the living animal contraction occurs most readily on closing the current, whatever be its direction. Fick and Orelli[6] found the same happen in their experiments on the ulnar nerve in man. Schiff[7] puts down as a law that "contraction occurs in the living subject on making the current in

[1] Hermann says (loc. cit. vol. ii. p. 63): "Pflüger the first showed that stimulation of the nerve occurs at one electrode only, on making the current at the kathode, on breaking it at the anode"; and recognises Chauveau's independent discovery of the same fact. Biedermann, by the way ("Uber die polaren Wirkungen des elektrischen Stromes im entnervten Muskel," 'Sitzungsberichte der K. K. Akad. d. Wissenschaften,' vol. lxxix. p. 289), has successfully demonstrated the polar effects on curarised muscle.

[2] 'Beiträge zur nähere Kenntniss des Galvanismus,' Jena, 1802.

[3] 'Essai sur les Phénomènes électriques des Animaux,' 1840.

[4] Cf. 'Traité des Phénomènes electro-physiologiques,' chap. ix., "Usage thérapeutique du Courant continu." 1844.

[5] Loc. cit.

[6] 'Wiener Medicinische Wochenschrift,' 1856.

[7] 'Lehrbuch der Nervenphysiologie,' p. 80, 1856.

either direction, but no contraction on breaking it," adding, it is true that in his experiments he never closed the current for more than five seconds. Claude Bernard, whose classical lectures appeared at the same time,[1] says that "a nerve placed in normal organic conditions, fit to transmit voluntary impulses, gives only one contraction, which occurs on closing the current, whatever be its direction," adding that deviations from this law depended on the mutilation of the nerve.[2] This point has been lately taken up by Rumpf,[3] chiefly from a clinical point of view. He shows that the apparition of the A.O.C. is delayed by the influence of the central organs and hastened by the removal of this influence. There is thus a qualitative disturbance of the normal galvano-nervous formula, which has escaped notice hitherto, owing, probably, to the shortness of the period at which it is recognisable—a few days only, immediately after the injury. The subject deserves further investigation, but much care is required to avoid fallacies. It is probably owing to the influence of Remak[4] that electro-therapeutists have had so much difficulty in realising the full bearings of Pflüger's conclusions. Remak's researches on the effects of the galvanic current on the nerves in the living subject were mainly directed to the phenomenon of "galvanotonus," that is, of contraction during the continuous flow of the current. This he found to be more marked when the current was descending.

Grapengiesser, as early as 1801,[5] had expressed himself clearly to the effect that the negative pole was the more effectual of the two, both at the closing and during the passage of the current. Remak takes extraordinary pains to show that this superiority of the kathode, due to its chemical properties, exerts no influence on the results of the transverse stimulation of nerves. If, for instance, the two poles are

[1] 'Leçons sur la Physiologie du Système nerveux,' vol. i., 1858.

[2] Similar observations were made by Pflüger, Bezold, and Rosenthal. See Meissner's 'Bericht' for 1858. Romanes (Proc. Roy. Soc., 1876 and 1877) has measured the changes in the excitability of the frog nerve produced by injury. Thus, before section A.C. = 90; K.C. = 100; A.O. = 14; K.O. = 6; whilst after section A.C. = 140; K.C. = 300; A.O. = 195; K.O. = 14.

[3] 'Archiv f. Psychiatrie,' vol. viii. p. 567.

[4] 'Galvanothérapie,' 1859.

[5] Quoted by Remak, loc. cit., p. 102.

placed on the temples, it is noticed that contractions are more readily obtained on the side of the kathode; but this is probably due to the fact that the current there is "ascending," for the current avoids the resisting bones, and passes round about the forehead. Even were this not the case the phenomenon could be readily explained by the assumption that a nerve is more readily stimulated by a current flowing transversally through it from its inner to its outer aspect than in the opposite direction![1]

The method usually spoken of in electrotherapeutics as "Brenner's polar method" was, so far as its physiological basis is concerned, fully described by Baierlacher in 1859.[2] Before entering upon this subject, I may be allowed to offer a few remarks on the impropriety of the word "polar." By it is implied that the method rests upon a recognition of polar, instead of directional, influences, and is meant to distinguish it from the so-called "physiological" method. But the truth is, that, as we have already stated, in physiology, since Pflüger, it is to the specific action, chemical or otherwise, of each pole that the effects of the current are attributed. The physiological is, therefore, a polar just as much as the therapeutical method; the first is *bi*polar, the other *uni*polar. By these terms we simply mean that either both poles or only one pole is applied to the nerve. I need hardly point out that the term unipolar, taken in this sense, has nothing to do with the unipolar effects of induction discovered by Du Bois Reymond in 1845,[3] and since then studied by a large number of observers.[4]

In order to let the reader judge for himself how far Baierlacher had anticipated Brenner, I quote some abstracts:—

"The unipolar method of nerve-stimulation adopted by me gives us the opportunity of observing the separate action of each pole on the nerve, whereby we attain the peculiar conclusion that the same phenomena are produced which we are accustomed to consider as depending upon the direction

[1] Sounder views, however, prevail in Remak's later writings, e.g., in his 'Leçons sur l'Application du Courant continu.' Paris, 1865.

[2] 'Zeitschrift für rationelle Medicin,' series III., vol. v., p. 233. Compare Meissner's abstract in 'Bericht,' ibid., vol. vi. p. 442.

[3] 'Untersuchungen,' vol. i. p. 423.

[4] See Hermann, loc. cit., p. 86.

of the current. The positive pole on the nerve gives us the results of the 'ascending,' the negative of the 'descending,' current."[1]

The nerve chosen by Baierlacher for his first experiments was the peroneal, near the head of the fibula; and the result of numerous experiments was that,[2] "with the negative pole on the nerve, the closure contraction was very strong, the opening contraction absent, or exceedingly feeble. With the positive pole to the nerve, the opposite took place, the closure contraction being absent or very weak, the opening contraction strong." Thus the order was K.C.C., A.O.C., A.C.C., K.O.C.: a more accurate statement than that of Brenner, who places A.C.C. before A.O.C., owing probably, as we shall see, to a want of care in applying his electrode.[3]

The author further states[4] that "there can be no question about a difference in the direction of the currents" in his experiments, as the contractions appeared in the same order whether the indifferent electrode was placed above or below the other. He displays considerable acumen in the remark that, even with these changes in the position of the indifferent electrode, the upward or downward direction of the current can be but of secondary influence, compared with that exerted by the very different densities of the current at its points of entrance into, and exit from, the nerve respectively.

Baierlacher also repeated the experiment of Fick with both poles on the ulnar nerve, and came to the conclusion that the order of contractions was—

Current.	Descending.	Ascending.
Closure.	Very Strong.	Strong
Opening	Absent or Weak	Moderate

A result which coincides with the view that the anodal reactions of the unipolar method correspond with the "ascending," the kathodal reactions with the "descending," reac-

[1] Page 253. [2] Page 249.
[3] Or, at least, to his using a too large 'different' electrode. [4] Page 251.

tions of the bipolar method. This point is recognised by Meissner.[1]

Chauveau, at the same time, following an entirely different train of thought, came to similar conclusions: a result all the more meritorious that he worked in complete ignorance of all that was being done in Germany, and laboured under serious misconceptions, such as an imaginary influence of electrical "tension" and of "extra-currents." His experiments[2] were made with Leyden jars, induction coils, and galvanic batteries. The two former bring about contractions more readily at the negative than at the positive pole. On making a weak galvanic current, stimulation occurs at the negative pole only; the direction of the current has no influence whatever. For instance, when the poles are on the sciatic plexus and the lower part of the sciatic nerve, contractions occur in the thigh and leg when the cathode is on the plexus; in the leg only, when it is on the nerve.

The opening contraction occurs, under similar conditions, at the positive pole. In every case the indifferent pole may be applied to any part of the body without changing the results. Space does not allow me to indicate more than the general results of Chauveau's elaborate papers.[3] A good abstract of his researches has been given by Meissner, who adds[4] that these results coincide with those of Baierlacher,

[1] Loc. cit.

[2] "Théorie des effets physiologiques produits par l'électricité transmise dans l'organisme animal à l'état de courant instantané ou de courant continu," 'Journal de la Physiologie,' 1859, pp. 490, 553, 1860, pp. 52, 274, 458, 534, ff. See also Meissner's 'Bericht,' in 'Zeitschrift für rationelle Medicin,' 1860, p. 554.

[3] I cannot here pass under silence Chauveau's latest contributions to the subject of unipolar stimulation. ('Comptes Rendus de l'Académie des Sciences,' 1875, 1876, vols. lxxxi. pp. 779, 824, 1038, 1193, and lxxxii. p. 83). In a series of notes to the Academy he has formulated a number of propositions which are mostly in direct contradiction to all the received views, and his own former results. His recent experiments have been made both on frogs' and mammalians' nerves. To discuss them here is impossible, as they are but briefly described; but they will have to be carefully controlled. He reaches the astonishing conclusion, that for every motor-nerve there exists a current-strength (usually very weak) which gives to both poles the same degree of activity. Below it is the negative pole, above the positive, which preponderates. For sensory nerves it is the opposite that holds.—What precedes applies to the *closure* of the current. On opening also, Chauveau finds the positive pole the more active. His results are embodied in a number of tracings and tables of curves.

[4] Loc. cit., p. 455.

and with Pflüger's theory that stimulation is produced at the appearance of katelectrotonus (= closure contraction when the kathode is on the nerve), and at the disappearance of analectrotonus (= opening contraction when the anode is on the nerve).[1]

The untiring zeal and brilliant success of Brenner in applying the unipolar method to the practice of electro-diagnosis, his success in upsetting the obsolete views of Remak and his followers,[2] are quite sufficient to secure him a prominent place in the history of the question; though Pflüger's law, rightly understood, on one side, and the results of Baierlacher and Chauveau on the other, deprive him of the priority which seems generally to be conceded to him. It was only in 1862[3] that he first published his "discovery" that the closure contraction depended upon the negative pole, the opening contraction upon the positive. In his great work[4] he has collected a vast array of experiments and illustrations in support of his views. The first volume is devoted to the application of the unipolar method to the acoustic nerve, which gives a very pure series of reactions in the normal condition, responding to the cathodal closure (and duration) and anodal opening only. He endeavours to base a rational system of electro-otiatrics on this result. In the second volume he describes the apparatus and manipulations neces-

[1] I think I may fairly claim as supporting the main argument of this paper the results obtained by Morat and Toussaint ('Comptes Rendus de l'Académie des Sciences,' 1877, vol. lxxiv., p. 503), with reference to the electrotonic state of nerves excited on the unipolar method. These observers found that the modifications were of the same order on both sides of the point of application of the electrode. When the kathode was applied, the modification was positive at both ends; when the anode, the modification was negative. Now, in the usual bipolar application, the modification at each end is of the same name as the neighbouring pole. The authors explain the results of the unipolar excitation by saying that the current follows the nerve in two directions; opposed to the natural nerve current in one case, concordant with it in the other; hence the negative and positive modifications. But is it not simpler to assume that it is the virtual anodes or kathodes, on either side of the actual electrode, which produce the phenomenon? When the positive pole is on the nerve, the two virtual kathodes will naturally call forth the negative modification at each end; and the opposite will happen when it is the negative pole which is in contact with the nerve.

[2] See, for instance, Benedikt, 'Elektrotherapie,' 1st Edition, 1866, so unsparingly criticised by Brenner in his 'Untersuchungen,' vol. ii. p. 208, ff.

[3] 'St. Petersburger Medicinische Zeitschrift,' vol. iii.

[4] 'Untersuchungen und Beobachtungen auf dem Gebiete der Elektrotherapie,' Leipzig, 1868-69.

sary to operate successfully, discusses the relations of the "polar" to the "physiological" methods, and makes a substantial contribution to the art of electro-diagnosis.

The effect of Brenner's work was to call forth a controversy as violent as it was useless, but it stimulated also the zeal of a large number of workers in the field. Though the din and dust of the battle caused the loss of much time and temper, there gradually arose clearer views, and valuable results were obtained. The question of the possibility of demonstrating the phenomena of electrotonus in the living human nerve occupied the attention of several observers. First, Fick,[1] whose results were negative, then Eulenburg[2] and Erb[3] appear in the field. Eulenburg, applying the galvanic current to an accessible nerve, such as the peronæus, ulnar, &c., tested its irritability by means of an induced current below the point of application. His results were in accordance, apparently, with those obtained on the exsected frog's nerve; and he found increased or diminished irritability when he produced descending extrapolar katelectrotonus or anelectrotonus. Erb was not so successful in his first experiments, and found a complete inversion of the phenomena, though he followed a method similar to Eulenburg's. Helmholtz[4] thereupon made an important suggestion, which seems to me to contain, as in a germ, the solution of the main difficulties encountered in the experimentation on the living body, and which will be developed further on. He said that Erb's paradoxical results might be due to the fact of the electrical diffusion, which in the immediate neighbourhood of the electrode must be sufficiently considerable to justify the assumption that it there acts as an opposite pole. Acting upon this supposition, Erb tested the condition of the nerve immediately under the electrode, and found it coincide with that obtaining in physiological experiments. The results of Samt[5] are interesting

[1] 'Medicinische Physik,' 1866.

[2] "Ueber elektronisirende Wirkung bei pereutoner Anwendung des constanten Stromes auf Nerven und Muskel," 'Deutsches Archiv für klinische Medicin,' vol. iii. p. 117 (1867).

[3] 'Ueber elektrotonische Erscheinungen am lebenden Menschen,' ibid. p. 513; and 'Galvanotherapeutische Mittheilungen,' ibid., pp. 238 and 333.

[4] In an oral communication to the Naturhistorisch-Medicinisch Verein meeting at Heidelberg, 1867.

[5] 'Der Elektrotonus am Menschen,' Berlin, 1868.

chiefly on account of his conclusion that many of the inconsistencies among the results obtained on the human subject are due to morbid conditions of the nerves: a view supported by the fact that the gradual exhaustion of a frog's nerve is a frequent cause of fallacy in experiments, and that the ventral organs exert a marked influence in the time of apparition of the several reactions.

Brückner's results[1] do not throw much further light upon the subject; but the method he used suggested to Runge[2] a series of experiments which are described in a very able paper. This method consisted in intercalating in the circuit of the polarising current the secondary coil of the testing instrument. Brückner found that when the two currents moved in opposite directions the effect was diminished at the negative pole of the induced current especially. Runge performed a series of ingenious experiments, the result of which goes far to prove that in all anterior electrotonic experiments the summation of effects of the two currents must be the real cause of the phenomena, and not the altered irritability of the nerve. Ziemssen[3] coincides with this view, which he supports by experiments of his own. But he justly condemns the sweeping assertions of Runge, who, forgetting the results obtained in physiological experiments by irritating the nerve with chemical agents, would explain the whole question of electrotonus by a mere antagonism or combination between the polarising and testing currents.

The only experiments on electrotonus in the living man where due care was taken to avoid the innumerable sources of fallacies which beset all such attempts, are those of Cyon,[4] made in 1868. By dint of trouble and care, he obtained normal results in a *few* cases: a significant fact, and which, in itself, speaks volumes against the basing a therapeutical system upon the supposed electrotonic influences of the rough

[1] "Ueber die Polarisation des lebenden Nerven in Menschen," 'Deutsche Klinik,' 1868 and 1871.

[2] "Der Elektrotonus am lebenden Menschen," 'Deutsche Archiv für klinische Medicin' (1870), p. 356.

[3] 'Die Electricität in der Medicin,' 4th Edit., pp. 63 ff.

[4] 'Principes d'Électrothérapie,' Paris, 1873. At the same time I fully agree with all that Erb says of this work in his severe review of it. (Virchow's 'Jahresbericht' for 1873.)

and ready proceedings usual in medical applications of electricity. Cyon experimented on the ulnar nerve; he found it necessary to fix the arm in a plaster of Paris mould; applied the polarising electrodes over points where the nerve was superficial, and tested the extrapolar descending electrotonus by means of induction shocks. The electrotonic condition was expressed in terms of the contractions of the adductor pollicis; their amplitude being registered on a revolving drum by means of a lever attached to the thumb. The cases in which he was unsuccessful he explains partly by morbid conditions of the nerve, partly by the influence of the central organs; but apparently ignores Helmholtz's explanation, which is of far greater importance, that the disturbing element lies in the irregular diffusion of the current from the nerve among the surrounding tissues.

Hitzig[1] in a valuable contribution, unfortunately unfinished, among other points discusses the reactions of the acoustic nerve to the galvanic current. It is the only nerve of the body on which we obtain reaction to anodal opening and cathodal closure *only*. The reason given for this fact by Hitzig is not only satisfactory, but most suggestive. The deviations from the typical formula, he says, occurring in unipolar excitation of other nerves, is due to their being surrounded by good conductors, as suggested by Helmholtz, with reference to Erb's experiments on electrotonus. Now the acoustic nerve is surrounded by bone, a bad conductor, and ends in a substance homogeneous to itself; therefore the whole of it may be thrown into a state of anelectrotonus or katelectrotonus: hence the purity of its reactions. I fully agree with Hitzig in his various strictures on Brenner's views; but think with Erb[2] that it is a fallacy to argue to the absolute non-existence of polar effects from the inefficacy of transverse electrisation of the nerve. May it not be assumed that under such conditions the two opposite polar actions are in equilibrium and neutralise one another?[3]

[1] "Ueber den relativen Werth einiger Elektrisations Methoden," 'Archiv für Psychiatrie,' vol. iv. p. 159 (1874).

[2] "Ueber die Anwendung der Elektricität in der inneren Medicin," 'Volkmann's Vorträge,' No. 46.

[3] The possibility of transverse stimulation of nerves is at present the subject

Filehne[1] has laboured very hard to reconcile the "therapeutical" and "physiological" methods. Considered from a general point of view, such a reconciliation appears unnecessary: impossible indeed from a more special standpoint, since the very essence of the bipolar method consists in acting on the isolated nerve with the two poles at their full undivided power; whilst that of the unipolar method consists in eliminating, as much as possible, by scattering it, the effect of one of the poles. Pflüger having shown that contraction depends on purely polar effects, the problem was rather to explain how it is that with the unipolar method the anodal closure usually preceded the anodal opening contraction, and how anodal closure and cathodal opening contractions occurred at all. Filehne has done this but very imperfectly. He shows that in unipolar excitation the current diffuses both up and down the nerve; and that the results were as if half the neutral pole was placed above, and half below the active pole. We readily grant this, but then are at a loss to understand how he ever could obtain results comparable to those of the bipolar method, as he never opposed but the inferior half of the divided to the whole undivided pole. In fact, his experiments repeated by Burkhardt[2] led this observer to different results. As to his experiments with excessive currents—8–30 cells to a frog's nerve, 50 cells to a rabbit's!—in which he obtained anodal make and kathodal break contractions only, we may safely assume that this phenomenon was rather due to injury to the nerve from the powerful chemical action, and hardly adducible as corresponding to Pflüger's third stage. At any rate Burkhardt did not find it at all constant in its occurrence. It must be noted, by the way, that Filehne does not mention how far he confirmed his data—apparently obtained from very few experiments—by an extended series of controlling tests, a very necessary precaution in this field of inquiry. It has also to be proved that the greater irritability of the upper part of the

of a controversy between Tschiriew, who has affirmed it ('Archiv f. Physiologie,' 1877 and 1879), and Hermann, who denies it ('Handbuch,' 1879, vol. ii. p. 80).

[1] "Die electrotherapeutische und die physiologische Reizmethode," 'Deutsches Archiv für klinische Medicin,' vol. vii., p. 575, 1870.

[2] 'Physiologische Diagnostik der Nerven Krankheiten,' 1875.

exsected frog's nerve is not the result of mutilation, before assuming, as he and others have done, that the anodal stimulation corresponds to the descending, and cathodal to the ascending current. Baierlacher and Meissner, as stated above, held an opposite view.[1] Filehne's views of a "peripolar" stimulation, it may be added, remind one very much of the experiments of Rousseau, Lesure, and Martin Magron,[2] in illustration of the fallacies in the results of Longet and Matteucci, due to the existence of derived currents in the undivided nerve. Chauveau[3] had also shown that the effects of lifting out the nerve on the electrode, and so exciting it by a true "peripolar" current, whilst the other was applied to the limb, were the same as those of unipolar excitation of the nerve *in situ.*

In much of what has been written on the subject of unipolar stimulation, there seems to me to underlie a fundamental fallacy: I mean a confusion between positive and negative *potential*, and positive and negative *pole* (or anode and kathode). It is quietly assumed that when, for instance, an electrode is held in each hand, one of the arms is under an anodal, the other under cathodal influence; but the fact is, they are simply at potentials different, positively and negatively, from that of the earth.[4] It cannot too strongly be dwelt upon that potential means simply *level*, and differences of electrical potential differences of electrical level, without which a flow of electricity is impossible. But by anode and kathode we mean a totally different thing; we mean the point of entrance of the current into, and its point of exit from, an electrolyte, that is, a conductor in which electrolysis is set up. Further, in a circuit composed of several electrolytes, the boundary between each pair of electrolytes acts as kathode to the one which the current leaves, and as anode to the one which it enters.

It is clear then that in sending a current through a composite electrolyte, such as the human body, there can be no question of exclusive localisation of "polar" effects. At every

[1] See Heidenhain, 'Meissner's Bericht,' 1857, p. 420.

[2] 'Gazette Médicale,' 1858; see also Bernard's 'Leçons sur la Physiologie du Système nerveux,' vol. i. p. 180.

[3] Loc. cit. [4] De Watteville, 'Introduction to Medical Electricity,' p. 111.

point where the current passes from one liquid, one tissue, or one cell, into another, there is an anode and a kathode, each endowed with its full chemical and physiological properties. Let us consider what takes place when the positive electrode is applied to the skin over a nerve. The current passes through the epidermis and subjacent layers, and part of it enters the nerve-fibres; at this point we have what I call the *virtual anode.* The greater portion of the electricity which has entered the nerve leaves it almost immediately, on account of the better conductivity of the surrounding tissues: these points of emergence form the *virtual kathode* of the nerve. The relative density of the current at the virtual electrodes depends upon its diffusion; and this again is regulated entirely by physical conditions, viz. the relative position of the actual electrodes, and the relative conductivity of the nerve and surrounding tissues.[1]

Now, it is upon the action of the virtual electrodes that the phenomena of electrotonus, and hence of contraction, depend; and this consideration is, in my opinion, capable of explaining all the apparent anomalies, without any reference to a "peri-polar" direction of current, or any such hypothesis. The appearance of katelectrotonus is a more powerful stimulant than the disparition of anelectrotonus. Hence when the positive pole is over the nerve, though the virtual anode is denser[2] than the virtual kathode, the latter will overtake the former, and produce a closer contraction, equal to or stronger than the opening contraction, as soon as $\frac{D^-}{D^+}$ is equal to or greater than $\frac{S^+}{S^-}$ (when D. and S. stand for density and stimulating energy of anode and kathode respectively). Hence also the great preponderance of kathodal closure over kathodal opening: the latter labours under double disadvantage, since the virtual anode now, besides its inherent inferiority, is not sufficiently dense to exert much influence. Brenner, in

[1] If a nerve lie isolated at any point of its course, and placed in contact with an electrode whilst the circuit is closed at any part of the body, the virtual electrodes of the opposite name will be at the points where the nerve emerges from the surrounding tissues.

[2] And hence more effective: Du Bois Reymond's law.

presence of the kathodal opening and anodal closure contractions, tried to explain them away by a mutual "overtaking" (übergreifen) of both poles.

In proportion as we, by the position of the actual electrode, can increase the difference of density between the two virtual electrodes we obtain purer polar effects. It is in this sense that Hitzig's explanation of the acoustic reactions must be taken. With most nerves of the body it is impossible even to approximate the normal formula; but in a few cases (such as with the ulnar at the elbow and wrist, and the peroneal at the head of the fibula) we obtain results which go far to prove my theory. If, for instance, one pole, being as usual on a distant, "indifferent" part of the body (the back, the sternum, &c.), the "differential" electrode is applied over the ulnar, close to the wrist, we obtain a series of contractions, K.C.C., A.O.C., A.C.C. (K.O.C.). Here we remark that A.O.C. > A.C.C. as it should be; but if we push the electrode a little higher up, where the nerve is immediately surrounded by good conductors (muscle and blood-vessels), we find A.O.C. = A.C.C., and finally A.O.C. < A.C.C. The explanation of this fact, according to me, is simply that in the first case the virtual kathode is spread over a greater length of the nerve, hence not so dense and effective; in the second, the current leaving the nerve immediately after it has entered it, the virtual kathode is, though not so dense as the virtual anode, still sufficiently so to make up the difference by its physiological preponderance.

Brenner gives synoptical tables intended to prove the parallelism between the "physiological" and his "polar" method; but with the exception that he shows that "apparition of katelectrotonus" is synonymous with "kathodal closure," and "disparition of anelectrotonus" with "anodal opening," these tables are useless. It would be better to tabulate the different conditions obtaining in the two methods. For instance:—

Physiological or Bipolar.	*Therapeutical or Unipolar.*
The nerve is mutilated and exposed, in direct contact with the electrodes. It is separated from its centres, and from its blood-supply.	The nerve is entire and lies in its physiological and anatomical relations, and is separated from the actual electrode by the skin and other tissues.
The actual and virtual electrodes are one, and the current at each usually of	The current at the virtual electrodes is of different and unknown densities;

equal and known density. They are placed one above the other.	the whole of one virtual electrode may roughly be said to be between the two halves of the other,
The full electrotonic effects, both stimulating and inhibitory, are produced at each electrode, and the law of contractions is based upon (1) the greater stimulating power of appearing katelectrotonus, (2) the interference of appearing anelectrotonus and of the negative variation of disappearing katelectrotonus.	The electrotonic effects are proportional to these densities, and the order and time of contractions depends upon (1) the greater stimulating power of katelectrotonus, (2) the relative density at the virtual electrodes.

In both instances the condition of vitality of the nerve will of course have to be considered as influencing the results. In experiments on the prepared frog's nerve it has long been noted that numerous circumstances, some connected with the process of exsection, others independent from it, exerted a disturbing influence on the results. In many cases these circumstances may be explained on the grounds that whereever the homogeneity of the nerve-fibres is destroyed a secondary anode and kathode exists. Even points where a considerable nerve-branch is given off by a nerve may set up such secondary electrodes, owing to the sudden change in the diameter of the nerve, and perhaps in the relative distribution of the connective tissue, which, of course, is to be looked upon as a different electrolyte from the nerve-fibres.[1] Again, in arguing from unipolar experiments on frogs, a possible source of fallacy may be found in the difference between the relative conductivity of nerve and muscle (and other tissues) in that animal and that obtaining in man: a condition which would materially alter the relative densities of the virtual anodes, under apparently similar circumstances. Any attempt then, I repeat, at *reconciliation* between the unipolar method of the electro-therapeutist and the bipolar method of the physiologist is, strictly speaking, meaningless, for there is nothing to reconcile; they start from the same fundamental axiom. The moment the different conditions under which they act in each case are explained the whole question is as clear as we can wish. In the bipolar method there is an element which does not exist in the unipolar: that of the *inhibitory*[2] effect of the lower pole on the influence of the upper pole. This effect

[1] Hering ('Sitzungsberichte d. K. K. Akad. d. Wissenschaften,' lxxix.) makes similar observations with reference to muscle.

[2] 'Sit venia verbo!'

comes into play only in the third stage of Pflüger's law, where the incipient anelectrotonus arrests the katelectrotonic impulse at the closure of the ascending current, and the negative modification arrests the anelectrotonic stimulation at the opening of the descending current. Trying to find anything similar when the unipolar method is used is a contradiction in terms, a begging of the whole question, and we have seen that Filehne's experiments on the subject are open to grave suspicion.

With reference to the experiments on the production of electrotonus in the human subject, it may be fairly said that they have led to very scanty results, and are exposed to a fundamental objection: that of the summation of the effects of the polarising and testing currents. But do we need any such experiments in order to estimate the electrotonic condition of any nerve? Is not the occurrence of opening and closure contractions, the best proof of the existence of anelectrotonus and of katelectrotonus? Taking the occurrence, then, of these contractions as an index to the condition to the polarised nerve, the obvious conclusion is that it is very rarely indeed, and on a very small scale, that we can ever produce anything like a pure anelectrotonus. In almost every position of the actual positive electrode, we obtain A.C.C > A.O.C.: a proof that the virtual kathode overpowers the virtual anode, that the katelectrotonus predominates over the anelectrotonus. The practical conclusion from this fact is that a therapeutical system built on the opposite anelectrotonic and katelectrotonic effects rest upon an imaginary basis. In by far the great majority of cases we can produce only predominant katelectrotonus; that is katelectrotonus with a more or less considerable admixture of anelectrotonus in its immediate neighbourhood. When to this consideration we add the fact that anelectrotonus immediately passes, on breaking the current, into a phase of increased irritability (positive modification) it is difficult to understand the precepts so often given that "the positive pole acts as a sedative, the negative as a stimulant." Both are stimulants, if "stimulation" there be, the kathode more so than the anode; but that is all, and I demur to the charge of inconsistency brought by Erb against those who,

adopting the unipolar method for purposes of diagnosis, do not carry it out systematically in therapeutics. A further objection to the electrotonic system of electro-therapeutics is that in the only instance where we can produce pure polar effects, in that of the acoustic nerve, "the systematic production of an- or katelectrotonus," as it is called, has not proved a success. Indeed, if the doctrine were true, we might expect that where the negative pole is indicated the positive would increase the mischief, and *vice versa.* It is a matter of daily experience that hyperæsthesic, hyperalgesic, and hypercinesic symptoms are, as a rule, happily influenced by the one pole as by the other. As to the pretension of inducing electrotonus of the brain, spinal cord, and vaso-motor nerves (katelectrotonus of the latter, by the way, to produce *dilatation* of the arterioles), I do not know upon what physiological evidence it rests, nor even what the expression very well means.

Though the unipolar method does not fulfil therapeutically the ambition of its promoters, its adoption has led to most valuable results in the field of diagnosis, and I am the more anxious to recognise Brenner's durable services in this respect that I have been led to stand in antagonism to his other views. Electro-diagnosis, difficult enough on this unipolar system, is impossible on the bipolar method, with which in the living subject we would get *four* electrodes to the same nerve; and the bitterest foes of the new system have been obliged to adopt it, though not always with the best grace.[1] A clear conception of the physical conditions of unipolar stimulation is, however, necessary for the rational application of electrical tests, and I am not without hope that the previous remarks may clear up certain obscurities hitherto prevalent.[2]

The question of a possible influence of the direction of the current is intimately bound up with that of the density of the current. On this point, again, much useless discussion has been expended. Supposing it is desired to estimate how much electricity passes through the sciatic nerve at a point half

[1] Cf. Benedikt, 'Elektrotherapie,' 2nd Ed. 1874.

[2] When the electrode is placed on the abductor indicis near its insertion, I find that A.C.C. > K.C.C. The same phenomenon occurs in other parts, and is explainable on the assumption that a virtual kathode occurs at a point of the muscle more excitable than the point of application of the anode.

way between the glutæal region to the popliteal space when the electrodes with a current of say twenty millevebers [1] are placed in these positions, all we shall have to do is to divide the diameter × specific resistance of the nerve by the diameter × mean specific resistance of the tissues of the thigh. The equation $\frac{D \times R,}{D^1 \times R^1} = \frac{X}{20}$ gives us the result. Assuming $D^1 = 12$ and $R^1 = 5$, when D and R are taken at unity this would give us $\frac{1}{60}$th of the current as passing through the nerve at the point, which is a very high estimate. This rough calculation gives us at least as accurate quantitative results as the galvanometric experiments of Burkhardt [2] and others, since the unknown relation between the resistance of the piece of nerve included between the needles and that of the galvanometer used can hardly be determined. This is not the place to discuss the arguments adduced by those who uphold the influence of the direction of the currents on the various tissues.[3] They cannot escape the objections deduced from the fact of a peripolar direction of the current in the nerve and from the great diffusion of the current in the interpolar region; it is only within a short radius of each electrode that the density is sufficiently great to produce physiological effects. And though the therapeutical conditions drawn from the electrotonic influence of the poles are not justified, it has yet to be shown that the very weak currents which traverse the tissues at a distance from the electrodes have any curative power, apart from any demonstrable physiological action. This supposition is, of course, within the range of possibility; but the partisans of direction-influence have hitherto produced no proof that their results do not depend from the relative position of the poles. The discussion of this point will be best deferred to another occasion.

If these pages, as I hope, have shaken one of the many *Idola Specûs* of electro-therapeutics, and disposed of some of the loose talk and thought so rife in the literature of the subject, my object is fulfilled.

[1] De Watteville, 'Practical Introduction to Medical Electricity,' chap. i. A current of 20 millivebers would be given here by about 40 Daniells.

[2] "Die polare Methode," 'Deutsches Archiv,' 1870.

[3] Onimus and Legros, 'Traité d'Électricité médicale,' Paris, 1872.

ANATOMY AND PHYSIOLOGY OF THE CHORDA TYMPANI NERVE.

BY HORATIO R. BIGELOW, M.D., WASHINGTON, U.S.A.

A SERIES of experiments, extending over a number of months, may, perhaps, give me a right to be heard upon the question of the "Anatomy and Physiology of the Chorda Tympani." It was not without much diffidence and a measure of embarrassment that I published, in June, 1879, in the 'Archives of Medicine' (New York), the conviction which I had reached that the chorda tympani is not a branch of the facial proper, but the continuation of the intermediary nerve of Weisberg, and that its physiology differed essentially from that claimed for it by writers upon special subjects of scientific medicine. I felt that I stood alone in a large field already worn by the onward march of physiologists, and that while their footprints all tended in the same direction, mine were bending in a line entirely opposite. But now that Dr. E. C. Spitzka ('New York Medical Record,' Jan. 31st, 1880) confirms my views in greater part (his objection to my theory of the function of the ganglion will be noticed farther on), I feel that a perfect vindication of such discoveries is merely a question of time. Prof. Sappey ('Traité d'Anatomie Descriptive'), than whom few write more clearly and forcibly, and whose opinion will always command profound consideration, writes: "La dissection unie à l'emploi des réactifs démontre entre la corde du tympan et le lingual une fusion intime, complète *fibrille à fibrille* dans toute l'étendue de l'adossement de ces nerfs; à l'aide de ce procédé on tenterait donc vainement de reconnaître le mode de terminaison de la corde du tympan." It

was this view of its anatomy which first led me to inquire most thoroughly and honestly into the relations of the chorda tympani, and, while my deductions are opposed in every particular to those of Prof. Sappey and of neuro-physiologists, yet may they be kindly received, and substantiated by individual investigation. In the 'New York Medical Record' for Jan. 17th, 1880, I published the following conclusions:—

1. The chorda tympani is distinct and integral throughout its entire length.

2. It is derived from the nerve of Weisberg, and not from the facial.

3. Its especial sensory function is derived from the ganglion upon the nerve of Weisberg, into the granular protoplasm of which the ultimate fibrils may be traced.

4. The lingual branch of the fifth presides over general sensibility only. Isolation of the chorda tympani destroys the sense of taste in the anterior two-thirds of the tongue, the fibres undergoing degeneration.

5. Section of the lingual destroys sensibility, but only modifies the sense of taste; this modification being due exclusively to the branches from the chorda tympani.

6. Section of the facial, behind the origin of the chorda tympani, destroys the sense of taste *only after a lapse of time;* and this not because the facial at this point contains gustatory filaments, but because the nerve is cut off suddenly from its supply, and has received such a shock that it undergoes degenetion. If the chorda tympani be drawn out at the point where we first notice its filaments of origin, and divided, the sense of taste will be almost entirely destroyed. If the nerve of Weisberg be cut in the aqueduct behind the ganglion, the sense of taste is lost. From which it may be inferred that the intermediary nerve is continued in the chorda tympani, and that this latter is a carrier of the sense of taste from the cells in the intumescentia gangliformis.

The differentiation of the intermediary nerve and the main trunk is attended with the utmost difficulty, and can be effected only by the use of reagents, "teasing" all the fibrils under a good lens mounted upon a tripod. In this way the chorda tympani may be separated from the lingual and followed

up: the terminal filaments expanding into the taste cells of the beakers, the bulbous expansion at the end being identical with the transparent nuclei. In the same way, by using a power of 1000 diam., we may trace into the intumescentia gangliformis the nerve fibrils of the intermediary nerve in the processes of the ganglion at their junction with the cell, and these may be traced into the granular protoplasm. From Dr. Spitzka's note in the 'New York Medical Record' of January 31st, I may be allowed, for purposes of discussion, a liberal quotation:—

"I am able to add an important confirmatory observation to those published by Dr. Horatio R. Bigelow in the 'Record,' favouring the view that the chorda tympani nerve is, after all, not a branch of the facial proper, but the continuation of the *nervus intermedius* of Weisberg. It is well known that this latter nerve is entirely distinct from the facial at the origin from the medulla oblongata. In numerous transverse microscopic sections of human and animal peduncular tracts, I have found that the fibres of the nervus intermedius have *no* connection with the facial nerve nuclei. Their central termination lies, in fact, in lower altitudes; that is to say, while the facial nuclei are situated within the lower pons margin, the nucleus of the nerve of Weisberg lies strictly within the limits of the medulla oblongata in the level of the superior auditory nucleus. The gray origin of the nerve of Weisberg is diffuse, and, in the *strict* sense of the term, cannot be called a nucleus for that reason. On examining its relations more closely, we find that it *does not correspond to the motor gray column* of the medulla, but to an ideal continuation of the gelatinous column of the trigeminus region; in other words, it originates in the *sensory* gray column of the medulla. . . . . Dr. Bigelow makes one statement to which I must take exception. He attributes the sensory function of Weisberg's nerve to a ganglion on the nerve. It is generally conceded that peripheral ganglia are obscure in their function, and that the function of a cerebro-spinal nerve is determined by its *central* connections."

This confirmation, in part at least, of my own views was most opportune. It was gratifying to be sustained in so

important a matter by one so skilled in investigation as Dr. Spitzka is known to be. Unquestionably it is a fact that the consensus of medical opinion concedes that the function of a cerebro-spinal nerve is determined by its *central* connections, rather than by peripheral ganglia. But such concession is due to the seduction of pleasant theorizing rather than to absolute demonstration. No one has attempted a logical explanation of the functions of the peripheral ganglia, and their existence upon sensory nerves seems to be ignored in the discussion of the characteristic nerve function. It may or may not be true that the functions of *all* cerebro-spinal nerves are determined by a *central* connection; we have no demonstrable proof for or against the theory. In all exact physiological inquiries, as, indeed, in all inquiries of exact science, we must be governed by fact rather than by inference. I doubt exceedingly if the chorda tympani is made sensory solely through its central connection. I can make no absolute assertion, because experiments upon this ganglion are surrounded with almost insurmountable difficulty. I have exposed it, and touched it with acetic acid; but a microscopic section showed that the chemical action had extended along the nerve fibre, and that it had lost its normal characteristics. I then tied the nerve with a small silk thread behind the ganglion, and irritated the ganglion with the electrode of a small Gaiffe battery; the sense of taste persisted for two hours and a half, so that there must have been reserve force at least within the ganglion. If it were possible either to extirpate the ganglion, or to destroy it without resultant injury to nerve fibre, the question might be definitely settled; and to this end I have called to its fullest extent upon the little inventive genius I may possess, but so far unavailingly. I do not maintain that the intumescentia gangliformis in question is capable of originating the sensory function, although in some cases of paralysis it would seem to possess such a power; but one of my experiments has demonstrated that it does act either as a storehouse or as a generator. The ganglion, too, serves to differentiate with exquisite precision between the effects of sapid substances, whose appreciation is governed by a central connection; the psychic phenomena of the cord

contrasting strongly with the physical manifestation of the ganglion.

In closing this paper, I may be allowed to refer once more to my communication in the 'Record.' "The difficulty of noting the impairment of taste is exceedingly great, and one will fail often before arriving at satisfactory results. The shock following a section of the nerve within the cranial cavity necessarily modifies exact conclusions, and, as Flint very justly observes ('Text-Book of Human Physiology,' p. 761), 'We must remember the difficulty of such observations, and we are to rely mainly upon the unmistakable phenomena noted in cases of affection of the chorda tympani in the human subject.' Paralysis of the facial, behind the origin of the chorda tympani, is attended with loss of taste in the anterior two-thirds of the tongue. But this is not due, as I believe, to the existence of gustatory filaments in the facial itself, but to the fact that the transmitted shock is so great as to destroy the function of the ganglion upon the nerve of Weisberg, and in this way inhibit the special characteristic of the chorda tympani as a carrier of the sense of taste. The sensibility of the tongue remains, because it derives its sensory power from a different source; the cells furnishing the lingual branch of the fifth are not interfered with in such cases of paralysis. It is a fact, however, that sensibility is often modified, owing to the inability of the nerve fibre, by reason of a degeneration resultant upon shock, to carry the sensation. If we isolate, so far as it may be possible to do, with a proper regard for the life of the animal, both the chorda tympani and the intermediary nerves, the sense of taste becomes greatly modified, and after a time almost entirely lost, the feeble sense remaining being due to the fibrils which it is impossible to divide. If there were essential gustatory filaments in the facial, the sense of taste would persist in a greater degree than it does after division of these two nerves. If we are to study the functions of the chorda tympani from its action in those morbid conditions affecting it, the nature of the *shock*, as well as the resultant influence engendered by contiguity of structure or by actual contact, must never be lost sight of. Nerves, themselves uninjured, may suffer in their action by transmitted influences."

ON THE PARALYSIS WHICH RESULTS FROM ANGULAR CURVATURE OF THE SPINE.

BY JOHN DUNCAN, M.D.,

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Cases of caries may be divided into two classes, of which the types are very distinct, although they shade into one another with an infinite gradation. In the one class are those in which the disease has a constitutional, in the other those in which it has an external etiology.

In the spine it appears to me that this distinction is as distinctly marked as in other parts of the body. Patients of the first group have the strumous or tubercular diathesis; near relations have had phthisis, or white swelling, or glandular inflammations. They have themselves the general characters of the diathesis, or have suffered from its pathological manifestations. They have spat blood or had pleurisy. They have scars in the neck, or tubercle in the lungs, or chronic joint-disease. In short, the personal or family history is bad.

In such cases the angular curvature is rapid in its formation. Psoas, or lumbar, or cervical abscesses form early in the disease. The patients sink from hectic or from amyloid degeneration of the liver and kidney. If paralysis make its appearance it is commonly from pressure on the cord, and is sometimes suddenly fatal in the cervical region.

Patients in the second class have generally some distinct history of serious injury. Their appearance is robust and healthy. The family and personal history is free from trace of strumous disease. Abscess is comparatively a rare occur-

rence. Paralysis, on the contrary, appears soon. Sometimes it precedes the projection of the spines, and is accompanied only by tenderness on pressure or percussion, or on the application of heat or cold. The patients die from the effects of the paralysis, and if there be no paralysis the disease runs an exceedingly slow course. While the strumous form attacks the child or the young adult, this variety is more common in middle or advanced life.

The paralysis is peculiar. It confines itself specially to the motor track of the cord. Sensation is rarely affected. The motor paralysis is often of the spastic form, and is sometimes very extreme. The lower limbs are most apt to become paralysed. The bladder and bowels retain their normal functions, or are only temporarily disturbed. Trophic changes also are infrequent.

I have been much struck by the distinct way in which all these peculiarities come out in the series of cases which I shortly append. They are those which have been resident in the wards under my care during the last two years, having been admitted for the purpose of opening the abscess or for observation. Many other cases of the strumous variety were out-patients during the same period, but I have not met with any other examples of paralysis.

I.—Jessie S., æt. 31, married, admitted 18th December, 1878. Sallow and thin. Her mother died of phthisis, and her father died young. She is very subject to indigestion. About 4 years ago she felt pain in the back after delivery of her second child. She was unable to carry it about, and has not been free from the pain since that time. Three months ago swelling appeared about Poupart's ligament, which was opened. There is prominence of the 11th and 12th dorsal vertebræ. A plaister jacket was applied. In June, 1879, the discharge, though slight, still continued, and there was little alteration in the general condition.

II.—Harriet M., æt. 23, single, admitted 19th June, 1878.

History. *Family:* Mother and two sisters died of phthisis. A brother had an abscess in his side, which has now healed.

Personal: Amputation of the third toe of the left foot was

performed a year ago, on account of caries. There is now caries of the metacarpo-phalangeal joint of the left thumb.

There is lumbar abscess, and projection, and pain on pressure over the 4th and 5th dorsal vertebræ.

The abscess was aspirated and a jacket applied. When last heard of, the patient was progressing favourably.

It would be tedious to enter into the history of common cases of which these may be taken as the types. In all the treatment was either aspiration or antiseptic opening of the abscess, with the use of the plaister jacket. I enumerate only and mention the salient points.

III.—Duncan C., æt. 22. Psoas abscess and angular projection of the 10th and 11th dorsal vertebræ. Has twice had inflammation of the lungs, and the present signs of phthisis are distinct. A brother had white swelling of the knee.

IV.—John L., æt. 23, admitted 7th November, 1879. Psoas abscess and angular curvature of 6th, 7th, and 8th dorsal vertebræ. He refers his disease to a fall downstairs, but it is clearly ascertained that the projection existed before the fall, although the pain was thereby increased. Some years ago he had abscesses in the right arm, which lasted several months. His mother died after child-birth.

V.—A. D., æt. 3, admitted January, 1880. Double psoas abscess and excessive curvature in lower dorsal region. Two cases of phthisis in the family. The treatment is by double extension.

VI.—Patrick D., æt. 31. Pale, thin, and strumous-looking. Lumbar abscess and angular curvature of 7th dorsal spine. Knows nothing of family history. No injury or previous illness.

VII.—William S., æt. 25. Lumbar abscess and projection of 7th and 8th dorsal vertebræ. No history of injury. A brother has scars in his neck.

VIII.—John N., æt. 15. Caries of 3rd and 4th cervical vertebræ. Father died of phthisis. In this case I opened a retro-pharyngeal abscess by the method advocated by Mr. Hilton and Mr. Chiene. Unfortunately it opened also into the pharynx a week afterwards, and consequently became septic. He comes in to show himself occasionally, and is

getting much emaciated from the discharge, which has now been going on for more than a year. He has also had several fits. The epilepsy, however, I believe to be connected with carie-necrosis of the right temporal bone in the mastoid region, which is considerable in extent.

In none of these cases was there the slightest paralytic symptom.

The following cases illustrate the other form of disease of the vertebral bodies to which I have referred.

I.—The first case which attracted my notice was that of a miner, æt. 23, whom Professor Saunders asked me to see in his ward. There was marked angular curvature of the lowest cervical and upper dorsal vertebræ, with an unusual amount of inclination to the left. He said he had been in the habit of carrying exceedingly heavy weights on the left shoulder, and on several occasions was conscious of having strained the parts. In fact, he felt assured that he had immediately produced the lump in one of his "lifts." There was considerable pain on pressure. He had spastic paralysis of the lower extremity to an extreme degree. The tendon reflex was enormously exaggerated, and he was unable to walk more than a few paces, even with assistance, on account of the spasmodic jerking of his limbs. I applied the plaister jacket, with jury-mast and elastic support for the head. He at once began to improve. He still after a year continues to wear the apparatus, and sends me word that he can go about as well as ever, but his legs, he thinks, "make him walk faster than he would otherwise do."

II.—John K., æt. 27. Admitted December 7th, 1878. Family and personal history good.

A year before admission he fell from a ladder while carrying a heavy load of lead. He injured his back, and thereafter wore a belt for two months before he observed the projection of his spine. The 5th, 6th, and 7th dorsal vertebræ are prominent. He has distinct loss of power in the legs, which he moves in a jerking manner. The patellar tendon reflex is increased. Sensation is normal. He had a plaister of Paris jacket applied. The pain and paralysis much improved when he was last heard of, six months after leaving. He then showed himself for renewal of the jacket.

III.—John D., miner, æt. 53. Admitted May 6th, 1879. The family and personal history are unexceptionable. He is not aware of any special injury, but says that he has often felt his occupation a strain on his back.

He has felt pain in his back for about a year, and noticed the projection of the 4th dorsal spine several months ago. For three or four weeks his legs have been getting weaker. He is now barely able to walk with assistance, and his legs have a great tendency to cross in doing so. The tendon reflex is much increased in every action. Sensation is slightly duller than normal in the lower limbs. The bowels are sluggish. A jacket was applied, but fitted badly, and during the ten days he was in the infirmary he rapidly deteriorated. At the end of that time he was quite unable to stand alone. A better jacket was adjusted, and he at once began to improve. He returned January, 1880, to ask whether he might resume light work. He could then walk without the least apparent impediment.

IV.—Henry B., æt. 13. The family history is good. There is no history of injury. The 3rd, 4th, and 5th dorsal spines project. The disease has been in progress for three years, but there is no sign of abscess. On the other hand, the spastic paralysis is so marked that he cannot walk even with assistance. He has had a jacket on now several months, but although the local pain is relieved the paralysis is steadily getting worse.

V.—Thomas B., miner, æt. 32. Admitted 6th Feb. 1880, a robust-looking man; family history perfectly good.

As a boy he pushed waggons in the mine by laying his head against them, and remembers straining his neck when he was seventeen so severely that the pain lasted for a year, during which he could not work. Since then the neck has been stiff. Six years ago he lost the power of his fingers, and shortly afterwards of his legs. The fingers are now nearly well. The legs have got gradually worse, and he is now quite unable to walk. He has on several occasions had difficulty in passing water. This was much relieved by the application of a blister to the nape of the neck. The actual cautery has been used three times, twice with decided but temporary

improvement in the paralysis. Sensation is not affected; he can move his limbs pretty freely in bed, but cannot support himself. Tendon reflex is increased. The neck is shortened, and quite stiff. There is no abscess. The plaister jacket was applied 9th Feb. 1880.

VI.—William B., miner, æt. 38. Admitted July, 1879. Family history good. No previous illness. He once broke his thigh, and has had several severe injuries to the back, but cannot attribute his present illness to any one in particular. He has had pain in the back for two years, but left off work only five weeks ago, when the doctor observed the prominence of the 4th dorsal spine. The patient walks with great difficulty, and has all the symptoms of spastic paralysis. The leg continues to twitch for a considerable time after the stroke on the tendon. A plaister jacket was applied, when he immediately began to improve. It was renewed in Jan. 1880, when the paralysis was found to have almost disappeared.

VII.—James McGlore, æt. 37, miner. Patient of Dr. Muirhead's. The family history is unexceptionable. Four years ago, just before his illness commenced, he slept out all night while intoxicated. For two years he had paralysis and pain in the back, but no projection. The 3rd dorsal spine is now prominent. The legs are constantly and rigidly contracted, flexed, and crossed. If the great toe be forcibly flexed the limb is involuntarily straightened, but soon recovers its abnormal posture. Two years ago he had some difficulty in urination and defæcation, but it speedily passed off. Double extension was used, and the actual cautery applied, but without benefit.

VIII.—W. M., æt. 38. Admitted Feb. 1880. The family and personal history is good. He has met with no serious injury. Pain in the back came on about six months ago, with projection of 12th dorsal spine. He has distinct spastic paralysis with increased tendon reflex. There is no abscess. A plaister jacket has been applied.

The following cases differ from the others in several important features, and may be regarded as links in the chain which unites the two classes together:—

I.—James H., æt. 42. Patient of Dr. Muirhead's. A brother

died of phthisis. The disease began four years ago, without known cause. Pain in and projection of the 3rd, 4th, and 5th dorsal spines were at once observed, and an abscess soon afterwards formed a little lower down, which still continues to discharge. In August, 1879, he began to feel loss of power in the limbs, and abdominal constriction. He has now a trailing gait. The muscles are flaccid, and without spasm. The reflex action is diminished. The patellar tendon reflex is slightly increased, especially on the left side.

II.—Patrick L., æt. 38, labourer. Admitted July, 1879. Family history good. He met with a severe injury to the back by falling from a ladder a year ago, and immediately afterwards a gradually increasing projection of the 5th dorsal spine commenced, followed shortly by an abscess a little lower down. There is no paralysis. The abscess was opened antiseptically, and he is now progressing favourably.

These cases, I think, mark very clearly the distinction I have drawn between the strumous form of the disease and that which has its most striking analogy in the chronic interstitial absorption of the neck of the thigh-bone, which in adults so often follows severe injury. It will be noted that in this latter variety, to which I specially wish to direct attention, there was in almost every case a distinct history of injury, or of such an occupation as throws great strain upon the vertebral column. In the strumous the disease may have its starting-point in an injury, but most often the history is an afterthought, or the projection is said to have come of itself. The other peculiarities of caries siccum, to which I have already referred, are also remarkably illustrated by these cases. Of all these features the most singular seems to me the frequency with which it is accompanied by paralysis. I have no doubt that my experience of these two years has been unusual. Dr. Saunders's patient, an Ayrshire miner, was so greatly benefited by treatment that the patients T. B. and J. D., also miners in the same district, were induced to come by the report of his case. Still it is plainly much more frequent in this than in the strumous caries.

It will be noted that the paralysis was almost exclusively motor. It is a feature of all spinal disease that special tracts

of the cord should be very accurately marked out by it. Whatever the explanation, it appears to characterise this consecutive paralysis as distinctly as the idiopathic. One can, to a certain extent, understand how the motor portions of the cord are most prone to become inflamed in consequence of caries of the bone. But it is more difficult to determine why the spastic form of paralysis should so greatly preponderate. In Dr. Saunders and Dr. Muirhead's patients the disease was severe and typical; in others it was unmistakable, though not so far advanced. The cases are clear examples of those secondary degenerations which Türck and Charcot have described.

The success of the treatment has been gratifying. Five cases have been sufficiently long under treatment to allow of a judgment being formed. Three of these have been practically cured, although they still wear the apparatus. In one case no benefit has resulted from the jacket. In another the jacket was not applied as it had previously been tried in Glasgow. For him I used the actual cautery without effect, and permanent extension in bed from head and feet, was applied, but not persisted in from the annoyance and inconvenience it occasioned.

It is plain that in this chronic ostitis, as in the same disease in other parts of the body, our local treatment is twofold, rest and counter-irritation. These cases illustrate the advantage that may be gained from both. The miner, Thomas B., was benefited on many occasions by blisters, and twice by actual cautery. Unfortunately the relief was not permanent. I fear that if counter-irritation be used alone this is the usual result, whether blisters, seton, or cautery be the means employed. To do much good they require persistence, and with persistence comes a time when their beneficial effect ceases. They must be combined with rest. Now rest for the spinal column is difficult to procure. It implies abrogation both of motion and of pressure. This may be attempted in two ways; by retention in bed, or by the use of apparatus. Until Mr. Sayre made known the value of the plaister jacket, the inefficiency of apparatus was such that recumbency was the recognised treatment for disease of the spinal column, at least in the

Edinburgh school. But if the objects aimed at by apparatus be attained, its other advantages are manifest; and I believe that they are in great measure so attained by the jacket. It does not take off perpendicular pressure so completely as recumbency does. If the jacket alone be used, the body telescopes gradually into it, and the plaister sinks *en masse* unless it be very accurately adjusted to the pelvis. If the disease be at or above the 6th dorsal vertebra, it is essential that the jury-mast be also applied, and the head attached to it by elastic bands which act much better than rigid straps. But while apparatus may not thoroughly relieve pressure, at least it very effectively restrains motion, which mere recumbency cannot. Even the actions of breathing are diverted from the ribs.

It is not essential that the material be plaister of Paris. That is usually most convenient. But I have moulded very comfortable jackets from the poroplastic splint material, and paraffin is light and easily applied in children, though not so durable and rigid in the adult.

The conclusions to be drawn from these cases are:

1st. That there are two distinct varieties of inflammation which attack the bodies of the vertebræ.

2nd. That in strumous cases there is comparatively little tendency to affection of the spinal cord.

3rd. That in chronic interstitial absorption there is a great tendency to paralysis, which presents the usual characters of what has been termed "pressure myelitis," with its secondary degenerations.

4th. That this paralysis may often be cured by rest and counter-irritation.

ON SOME VARIETIES OF CERVICAL PARAPLEGIA.

BY THOMAS BUZZARD, M.D.,

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It has happened to me to have under observation, more or less recently, several cases of the condition to which the term "cervical paraplegia" has been (first of all, I think, by Sir William Gull) applied. In these cases paralysis, usually both of motion and sensation, has affected more or less completely all four extremities, the upper in excess, and the circumstances by which the paralysis has been characterised have enabled one without difficulty to localise the lesion in the upper part of the spinal cord. In only one case, however, did the result give the opportunity of confirming the diagnosis by a post-mortem examination, and on this account I propose to relate this example first. The symptoms during life, and the pathological appearances after death, will serve to throw light upon many points in the other cases described, which have terminated in a greater or less amount of recovery. In spite of the aid thus afforded, it will be found that although the locality of the lesion can usually be indicated with tolerable confidence, its nature is by no means always to be determined with equal certainty.

Case I.—*Epileptic seizures—incomplete paralysis of arms and legs; pseudo-neuralgic pains; amyotrophy; marked improvement under iodide and mercury; relapse; apoplectic seizure; death.* Autopsy:—*hypertrophic cervical pachymeningitis; transverse myelitis; thickening of basilar artery; thrombosis; cerebral softening.*

The patient, S——, was a clerk, about 35 years of age, living a few miles from town, whom I visited at the request of his employer, on July 14th, 1877. He was below the middle

stature, with a squint, which I was told was of many years standing. He presented a peculiarly muddled look, and a manner so confused that I had difficulty in obtaining information from him. His memory appeared to be very bad. From what I could gather then, supplemented by later information, his family history was as follows. His father died at 38 years of age, in a lunatic asylum; his mother, æt. 50, of a "natural death;" she drank. He had never had any brothers or sisters.

Personal history.—The patient suffered from fits in his infancy until he was about six or seven years old. The next thing one learnt was that when he was 21 years of age he lost the sight of each eye in succession, that of the left eventually recovering. About eight or nine years ago he had a venereal ulcer, which was accompanied by two suppurating buboes, for which he took medicine to make his gums sore. His employer tells me that five or six years ago S——'s eyes were "bad" (interstitial keratitis?), so that he had to be off work for six months. During the last year he had had five or six fits, of which two occurred in the week preceding my visit. On the occasion of the first of these he felt dizziness, and was obliged to lay hold of his desk. He thought he was going to fall, and shortly afterwards he did fall, unconscious. He is not aware of having bitten his tongue.

Present state.—The sight of the right eye is almost nil, on account of a large leukoma of the cornea, concealing nearly the entire pupil. The squint, which is said to be of old standing, is not accompanied by double vision. He walks feebly and with a tottering gait. There is no power of grasping with either hand. This failure of strength, he says, has only taken place during the last four days, but for the last week he has been feeling increasingly feeble. There is no marked want of symmetry in the face. His tongue is protruded straight. The patient cries out every now and then with aching pains in the arms, sometimes in one and sometimes in the other. Frequently there is much pain over the brows and across the back. At times, he says, he suffers from pain all round the lower part of the head behind.

For some months past he has been liable to what he calls "rheumatism" in his limbs, and aching pains across the head

and loins. There is what he describes as "painful numbness" in both arms.

The heart and lungs are healthy. Appetite and digestion bad.

By the ophthalmoscope (under great difficulty, owing to unsteadiness, very small pupil, and some considerable haziness of the media) the retinal vessels in the left eye appeared tortuous, and the outline of the disc indistinct. The large opacity precluded observation of the right fundus oculi.

His landlady told me that six or seven weeks before this his manner had appeared very strange, and his memory had failed him to a large extent. It seemed that for a week past he had been taking small doses of bromide. I ordered perchloride of mercury and iodide of potassium.

A week later he came to my house, distinctly improved in intelligence. I noted that he held his head as though he had a stiff neck, and complained of pains at the back of the head and across the right ear.

There was a peculiarly helpless look about his hands. The excitability of the muscles of his arms was diminished in a marked degree to the faradic current.

Whilst examining him with the ophthalmoscope, I noticed that he was constantly swaying about, and frequently exclaiming suddenly as from the effects of pain. His arms would jerk, and he complained of sharp pains in them.

His temperature was normal. The urine contained no albumen or sugar.

After another fortnight's treatment with mercury and iodide, he was manifestly much improved. There had been no repetition of a fit. He had lost the pains in his arms, and there was but little pain in the head. The right leg was stronger. He described a feeling of tightness around the waist, which he had experienced for two or three days, but which was relieved by the action of the bowels. The mind appeared to be quite clear. By the ophthalmoscope the left disc appeared to be more distinctly defined, but the vessels remained tortuous. He was ordered one-sixteenth of a grain of perchloride of mercury with twenty grains of iodide of potassium three times a day, and twenty grains of bromide of potassium at bedtime.

On August 3rd the following note was made: "Three days ago he had a repetition of a feeling which he had not experienced for a year previously, and which then ushered in a fit. The feeling was of a whirling round in the centre of his head. He had a trifling amount of pain at the back of the head.

"His walking power improves daily. He walked to-day the best part of a mile, although there is still weakness, especially in the right leg.

"There is wasting of the dorsal interossei in each hand, but the fingers have regained warmth and colour (they had been cold and dead-looking). Their movements are, however, still very clumsy. This morning he lifted the ewer, which he could not have done three days ago.

"The electrical reaction in the muscles of the forearm is somewhat improved."

The following note of diagnosis was also made at this time: "One would think, from the powerlessness and wasting of the muscles, together with loss of electric irritability, that here was a case of pressure upon the anterior roots of nerves, probably from pachymeningitis."

Three weeks later the patient had gained strength; and was able to walk as much as three miles. The arms, too, had greatly improved. He had resumed his usual handwriting with a pencil, but there was too much clumsiness in the fingers for the successful use of a pen; nor could he pick up coins or pins. "It was only the fingers," he said, "which were still obstinate and had not regained their pliancy." The hands felt generally warm in the evening. The warmth of the bed seemed to soothe them. Contact with metal appeared to send them "all ajar." There was no pain in the head: his appetite was good and he slept well. He had been taking the iodide with a little iron.

On September 12 it is noted that the patient can walk three to four miles without fatigue. The grasp of the left hand is very imperfect, and there is still wasting of the interossei, as well as an "electrical sensation" in that hand. His gait is rather jerky and disorderly. He is somewhat restless at night. There is a sense of fulness, but no pain, at the back of the head. A few days after this he began to get more pain in the right arm,

especially from the middle of the upper arm down to the tip of the ring-finger. On examination, the ring and little fingers of the right hand were found to be clumsy, and unnatural in their movements.

September 20th.—There has only been a little pain, "almost what he might describe as a sympathetic feeling," in the left arm. But the fingers of the left hand are much more helpless and clumsy than those of the right.

"At this moment" (I note) "there is no pain in the right arm, but he has had several paroxysms this morning and many in the night. The pain is almost unbearable. It comes on very suddenly—a burning aching pain, 'as if the vein were filled with molten lead.' The day before yesterday the pain was so intense that he 'started up, and broke into a perspiration all over.' A paroxysm will last about half a minute. It seems to begin at the top of the arm, and it 'must go its course.' After the pain has left, a feeling of relief alone remains. He can lift his arm during the pain; in fact the exertion of the muscles seems to give relief. It is usually when the pain is going on in the right arm that he gets a little pain in the left. But he thinks (questioned) that he has sometimes a little pain in the left arm when none is going on in the right."

Examination showed that the excitability of the muscles of both arms to induced currents was much diminished, but not removed. There was still wasting of the interossei, but not, it was thought, so great as had been. He was unsteady on his legs, especially on the left.

A week ago he felt pretty well, and went by rail to the Crystal Palace. He undressed in my room for examination, and appeared to be very helpless; I had to button his clothes. By the ophthalmoscope, I found the retinal vessels of the left fundus very tortuous, but the outline of the disc distinct enough. I dilated the right pupil with atropine, but the opacity prevented any observation of the fundus. Three days ago he went and dined with a friend in the City (a distance of some six or seven miles). The following day was spent indoors.

The patient was now placed under the care of Mr. Harrison, of Streatham, whom I met in consultation on November 1.

S—— was up and dressed, seated in a chair. The arms were much atrophied, and the legs also, but to a less extent. He did not complain of pain in the arms, but occasionally I observed them to be jerked, and then he would cry out. When asked to explain the cause, he said that he had the cramp in them.

The left forearm showed hyperæsthesia to cold, but there was delay in receiving the impression of pinches.

There was paralysis of the sphincters, with ammoniacal urine.

The patient was put on a water-bed and ordered one-sixteenth of a grain of perchloride of mercury with fifteen grains of iodide of potassium three times daily; also some lactophosphate of iron.

November 17th.—I heard that there was complete paralysis of the intercostal muscles, and the breathing was entirely diaphragmatic. The patient was having epileptiform attacks every day. He was thought to be dying.

November 20th.—As the gums were beginning to be sore, the mercury was stopped and the iodide continued.

December 10th.—Mr. Harrison wrote that there had been a slight improvement. The intercostal muscles were acting slightly, and there was more power in the arms and legs.

About a week before his death, which took place on December 24th, he was removed into other lodgings. At this time he could just slowly and tremulously lift a handkerchief and give a very slight squeeze with either hand. The arms appeared to be equally weak.

For many weeks before the end he could not stand, and was indeed unable to lift his foot from the floor; but during the last few days of his life he could do this. Towards the close he became exceedingly irritable in temper.

On December 23rd, he was taken with insensibility and stertorous breathing, and died the following day, having never recovered consciousness.

The autopsy took place 52 hours after death, in the presence of Mr. Harrison and myself. Weather very cold. The body was extremely exsanguineous; a small bedsore on the sacrum. Much wasting of muscles was noted in the forearms and hands, not so much in the upper arms. The legs were thin, but did not appear so much atrophied as the upper extremities.

Head.—There was some slight opacity of the arachnoid membrane. The brain-substance generally was soft, putty-like in consistence, wanting in firmness. The left crus cerebri was almost in a fluid state of softening. The surface of the pons varolii, and also of the medulla oblongata, was softened. The basilar artery was thickened, and contained a thrombus. There was a large quantity of fluid in the ventricles.

Spinal Cord ; anterior aspect.—On ripping open the dura-mater from below upwards no change is noticeable until the upper dorsal region is reached, when this membrane is found glued together with the soft membranes, and the whole mass firmly adherent to the cord. With the finger one feels a hardness about the size of a bean at the left side of the cord at the lower point of the cervical enlargement. Here it is found that for about 12 millimetres there is complete adhesion together of both hard and soft membranes: thence upwards for nearly 4 centimetres the pia mater is much thickened and adherent to the cord. At this part the cord, on section, is found to be of the consistence of rotten cheese. The whole extent of this softening is about two inches longitudinally. Above this again, and for the remaining 12 millimetres or so of the separated cord, the substance feels firmer, if not of natural solidity.

Posterior aspect.—The membranes appear natural, until we come to about 16 centimetres above the pointed extremity of the cord, where the dura mater is found thickened and adherent to the soft membranes. Slitting these up, they can with some little force be separated from the surface of the cord, and then this latter, as well as the exposed surface of soft membrane, wears an aspect of erosion, and is pink in colour. It is evident that one is tearing through recent inflammatory adhesion. This adhesion is very close for about 4 centimetres, and thence upwards, continues, though not so firmly, till we come to the cervical enlargement, where there is (as on the anterior aspect) a matting together of all the membranes. On cutting through the part where the hardness previously described is most pronounced, we find that it is of almost cartilaginous quality in section, and measures ·5 cm. in thickness. The cord is nearly fluid at this point, and scarcely a trace of the pattern of grey matter is to be seen on section.

There was no disease of the vertebræ.

My friend Dr. H. R. O. Sankey, of the County Asylum, Prestwich, was kind enough to examine the cord for me, and his report is as follows :—

"Transverse sections of the piece of cord which was sent to me were very difficult to cut, owing to the friability of the cord substance and the toughness of the thickened pia mater.

"*Microscopical appearance of Pia Mater.*—At the posterior part of the cord this was thicker than at the anterior, and where it was thickest it measured about $\frac{1}{10}$ inch. It consisted of connective tissue, but was not at all uniform in structure. At some places it was highly cellular; at others chiefly fibrous. The arrangement was very irregular, but at one part an appearance was seen in which the fibrous bundles were very regular in arrangement and parallel to the cord. But few vessels exist in this structure; those observed, however, were well developed, especially as to the adventitia, which passed insensibly into the general fibrous matrix in which they lay. In one case a very thin-walled lacuna-like space was seen filled with blood, which was uniformly coagulated, and had not long been in this position.

"The band of connective tissue which passes into the anterior fissure is greatly thickened, and contains vessels which are altered in the same manner as those of the substance of the cord (vide infra). The adhesion between the pia mater and the cord substance was broken down in the cutting of the sections, and cannot be made out, but it is probable that the entering trabeculæ are not thickened, but rather softened, like the cord substance.

"*White Matter of the Cord.*—This is throughout greatly altered. The changes are as follows :—

"1. Ascending fibres are in many parts only represented by homogeneous globules, staining slightly in carmine and logwood; while in others a few more or less normal fibres are seen; and in others only the shrivelled sheath of Schwann can be seen, devoid of axis cylinder, and filled with a homogeneous drop of myelin.

"2. Connective tissue is greatly increased. As to its cellular constituents, immense quantities of new cells, darkly stained, cover the field of view at every part.

"3. A granular débris fills the spaces left by these two elements. (N.B. Some of these appearances may be due to post-mortem decomposition, i.e. the state of the more healthy nerve tubercles and the granular appearance just described; the cells cannot be.)

"Vessels are large, tortuous, and the walls are covered by new cells, so that their structure is in places quite obscured. In some places, where it can be seen, it is healthy, and the vessels are all void of blood, or nearly so, and patent.

"*Grey Matter.*—Of the two cornua (anterior) the left is the more diseased. In this one, two or three nerve cells can be seen. These are circular bodies, shorn of all processes, showing a granular nucleus evidently in the last stage of degeneration. In the right cornua a larger number of cells are seen. They are more refractive than usual. They are in many instances devoid of processes, but retain them in other cases for a short distance. The posterior cornua are involved in the disorganisation. It is, however, impossible to say that they have suffered more than other parts, or that the right has suffered more than the left.

"All that can be made of the rest of the grey matter is that it contains too many nuclei, and is granular and opaque, and contains vessels surrounded by cells to a great extent. One vessel near the central canal contains an ante-mortem clot.

"*Summary.*—1. Fibrous thickening of pia mater.

"2. Infiltration of cord by new cells from vessels, and increase of existing connective tissue by migration or otherwise.

"3. Destruction of normal constituents.

"4. Post-mortem decomposition."

I take this to have been a case of inflammation of the internal layer of the spinal dura mater, of the arachnoid, and pia mater, the hard and soft membranes being agglutinated at the point described into a mass which was strongly adherent to the cord. The external layer of dura mater appeared to be unaltered. Dr. Sankey speaks of the tough mass as thickened "pia mater," but I imagine it is difficult to say with which of the membranes the hypertrophy described is to be especially associated, for they are all involved.

Charcot,[1] writing of hypertrophic cervical pachymeningitis, remarks that it consists of an alteration of membranes affecting more especially the dura mater, and notes that the cervical enlargement of the cord is in some respects a favourite seat of it. The alteration in the dura mater is the primary fact, but later on the cord and the peripheral nerves are involved. He thinks that the cases formerly described by Laennec, Andral, and Hutin, under the name of "hypertrophy of the spinal cord," properly belonged to cervical pachymeningitis; the swelling, which was really due to the membranes, being attributed to the cord itself. As a matter of fact, the cord, far from being hypertrophied, is squeezed from before backwards. The pia mater is affected, but much less than the dura mater, which may attain a thickness, he remarks, of 6 or 7 mm. (In the case above described the thickness, when the cord was fresh, was 5 mm.) The dura mater is usually altered in its entire thickness, as is proved by the adhesions which unite it outside to the vertebral ligament, inside to the pia mater. In the present case it will have been observed that the external layer of dura mater was free from change.

With this remarkable thickening and agglutination of membranes the cord at the part most affected by meningitis was broken down and disorganised. The condition was one of meningo-myelitis, which I should think was probably syphilitic in origin, for the following reasons:—1. The patient had suffered some eight or nine years previously from a venereal ulcer. 2. The autopsy showed the basilar artery thickened, and thrombosed; the crus cerebri, pons varolii, and medulla oblongata softened. The patient's age was about 35—too early for the arterial change to be reasonably ascribed to senile degeneration. The thickening (endarteritis) was precisely of the character which we see so frequently in the cerebral arteries in the sequel of syphilis. 3. During life, as will have been observed, an extraordinary improvement took place in the patient very shortly after he began to receive specific treatment, especially as regards his lower extremities.

In view of the state of the cornea in each eye, and the history of fits in childhood, it seems quite probable that

[1] 'Leçons sur les Maladies du Système Nerveux.'

the patient may also have been the subject of congenital syphilis.

I was anxious to discover whether in this case a microscopical examination would disclose thickening of the walls of the vessels in the substance of the cord. It appears to be very likely that in cases of syphilis there is frequently such a thickening of the walls of the spinal arteries as experience has proved to be the case as regards the intracranial arteries. Such a thickening was found in the basilar artery in the present instance, and it appeared, therefore, at the least probable that a similar condition might have existed in the vertebral arteries or their branches. I regret that it did not occur to me at the autopsy (which was made under circumstances of some difficulty) to remove specimens of the vertebral arteries for examination. The hardening of the cord was not accomplished very satisfactorily, and Dr. Sankey had some trouble in his examination. He found that the vessels, as well in the substance of the cord as in the connective tissue dipping into the anterior fissure, were large, tortuous, and their walls covered by new cells, but in only one instance was he able to observe the presence of an ante-mortem clot. This, however, isolated as it is, is of great importance. We are still unable to say anything certain regarding the stage at which this thrombosis of a thickened vessel occurred, or as to the relation in point of time between the inflammation of the membranes and the myelitis. Nor does the clinical history aid us in any attempt at deciding whether the cord or its membranes was first attacked with inflammation.

The paralysis of the arms in this man was throughout much more marked than that of the lower extremities. This was doubtless because in the former case it was due in great part to disorganisation of the centres within the cord, in the latter partly to the result of meningitis, partly also to the interruption of nervous impulses caused by the slow compression in the cervical region. The appearances would lead to the inference that under the specific treatment the recent inflammation of the membranes in the dorsal and lumbar regions had very much cleared up. The treatment came too late to affect to the same extent the more or less organised thickening of the membranes in the cervical region, although it

is surprising to note the amount of improvement which did take place in the state of the arms. I do not feel able to explain satisfactorily the reason of this.

The pains, as they were described by this patient, were characteristic of a membranous lesion, causing irritation, especially of the posterior roots of the brachial plexus. As Charcot has pointed out, these pains may be met with in intra-rachidian tumours, in Pott's disease, and in vertebral cancer, as well as in hypertrophic cervical pachymeningitis.

The contraction of the pupils which existed here is well known as an accompaniment of lesions of the upper part of the spinal cord.

According to Charcot, epileptic attacks sometimes manifest themselves periodically in cases of compression-lesion of the cord. Although this association is comparatively rare, he has been able to collect five cases of this kind. In the present case I believe that there was optic neuritis, although I am unable to speak so positively on this point as would have been desirable. The difficulty of using the ophthalmoscope was very great even when the pupil was dilated, as the patient presented ataxic movements, swaying to and fro on the chair, and jerking his arms occasionally, as he was seized with a dart of pain; and on account of the haziness of the media. I could find no trace of gumma in the membranes of the brain, but it is always possible that a formation of this kind may have been absorbed under treatment. In view of the probable existence of optic neuritis and the certain thrombosis of the basilar artery and the cerebral softening, I do not think we can reasonably refer the epileptic seizures in this case to the compression of the cord in the cervical region.

Case II.—*Stiffness and aching of the right arm, followed, after three months, by loss of power in the left arm and leg; atrophy of interossei in the left hand; impaired cutaneous sensibility of right arm; tenderness on pressing 4th cervical spine; return of power in left limb, with more or less spastic rigidity and exaggerated reflex; family history of phthisis.*

A lady, æt. 26, married two years, never pregnant, was sent

to me in July 1879, on account of weakness and numbness in her limbs. She complained that her right arm was stiff and ached, the hand being numbed and tingling as though it had "gone to sleep." This had been the case since March last. Her attention had first been attracted by a change in her handwriting. There was nothing the matter with the right leg. About a week before I saw her, the left arm, which had been previously unaffected, had lost power, and continued feeble, without any such tingling or numbness as was experienced on the right side. At the same time, with this accession of weakness in the left arm, she found herself swerving to the left from weakness of the left leg. The left leg, like the arm, continued weak. If she held anything heavy in her left hand it trembled. The aspect of the limbs was unaltered, and there was no apparent wasting.

In the arms the reaction of the muscles to faradism was normal. In the lower extremities there appeared to be some diminution of excitability in the tibialis anticus group of each leg, but this was doubtful, as it was equal on the two sides. The electric current was felt much more strongly in the right than the left leg.

The patient complained of the lower part of her back aching in the morning on waking. She had never suffered from bad pain in the head, but at times had been troubled with a little neuralgia. She was thin and strumous-looking. On her father's side there is much consumption, six aunts and one uncle (paternal) having died of phthisis before thirty years of age. There is no history of injury. The catamenia are regular.

A month after her visit to me, on getting out of bed one morning she fell down, owing to a rather sudden increase of weakness in the left limbs. I saw her a day or two afterwards, when the left arm was very powerless, especially about the scapulo-humeral muscles. In walking she swerved always to the left. There was a difference in the sensibility of the two arms, a touch being felt more on the left than the right—the sensibility of the right being evidently dulled. If she attempted to hold a book in the left hand, it slipped through her fingers.

I found that deep pressure on the spine caused pain over the fourth cervical vertebra. In no other part of the spine was tenderness complained of when pressure was applied, and repeated examination always caused it at this point.

The recumbent posture and general attention to nutrition by food and drugs was the treatment adopted.

In October last her condition was noted as follows :—

The right arm is somewhat stiff, and there is want of perceptive power by the fingers and thumb. The grasp of this hand is appreciable, but weak.

In the left hand there is some slight power of grasp, though less than in the right, and no apparent affection of sensibility. In using the arm, as in attempting to hold a fork, there is trembling of the limb. There is some atrophy of the dorsal interossei in the left hand with over-extension of the first phalanges, and some atrophy also of the thenar eminence. The over-extension of the first phalanges causes the meta-carpo-phalangeal joints to project in the palm of the hand. The patient can touch the back of the head with the left hand, the movement being accompanied by tremor, and causing some aching in the arm. The right leg has continued to be unaffected. The left leg is still weak. In walking, this leg is stiff, and the foot is carried in a semicircle. She tends to walk on the inside of the foot. There is difficulty in dorsal flexion, the foot trembling when she attempts it. There is some increase of patellar tendon reflex in this leg. The general health is good.

I have to-day (March 5th, 1880) received an account of her present condition. The left hand, it is said, can be used perfectly well for all practical purposes, although it feels rather weak and inclined to tremble. The middle finger is still *en griffe*, but she thinks there has always been a tendency to that position. During the past winter she has suffered much from chilblains, and the fingers have not yet recovered their proper shape. The muscles of the arm ache if she extends it from the shoulder, and if she attempts to do her hair.

There is still a little lameness in walking, and the sole of the left foot has a tendency to scrape the ground.

At no time has there been any affection of the bladder or rectum.

In this case we may at least localise the lesion in the cervical region. The circumstance of the left leg as well as the arm being affected would imply that it is the substance of the cord (and not merely the membranes) in this region which is in some way involved. An affection of the anterior roots of spinal nerves alone might explain the powerlessness, and also the muscular atrophy in the left hand, but the affection of the left leg, and, still more, the rigidity in these limbs, pointing to secondary degeneration in the lateral column, appears to show that we must seek the cause of the paralysis and atrophy in lesion of the anterior cornua in the cervical region. The lesion is one which apparently affects especially the left side of the cord. There may be a tumour, or, what would be more probable, considering the family history, and the pain experienced when the fourth cervical spine is pressed, some vertebral caries with pachymeningitis, causing compression and some resulting myelitis.

In the case which follows, and which in certain respects presents several points of resemblance to the one just related, the conditions observed are consequent on an accident. The lad appears indeed to have "broken his neck."

Case III.—*Injury to cervical spine whilst diving; complete paraplegia of all four limbs; recovery of power, with spastic condition of limbs from secondary degeneration.*

George H., æt. 18, by occupation a gardener, whilst bathing on July 18th, 1878, dived and struck his head against the bottom of the river in six feet of water. He was unconscious for a few minutes, and on coming to himself felt great pain in the back of the neck (level of third and fourth cervical spines), and found that there was total loss of power in all four limbs. Both hands were clenched after the accident, he says, and for a week he could not open them, nor could he move his head "any way." He says that he could feel "when they washed him."

About a week from the time of the accident he began to recover power gradually, in the right leg and arm first.

In January 1879 he could just manage to stand with a chair; in March he could walk a few yards by himself; and in April could dress himself. Since May he has been able to walk by himself with the aid of a stick.

When he applied at the National Hospital for the Paralysed and Epileptic as an out-patient, on July 9th, 1879, I noted that the electrical reaction of the left deltoid muscle was greater than that of the right to faradism, and less to the interrupted Voltaic current. The electrical reaction of the muscles of the legs appeared to be normal.

The patient was admitted into the hospital in October last, when the following note of his state was taken by Mr. A. E. Broster, Resident Medical Officer:—

"The spinal column shows nothing abnormal.

"The head can be moved perfectly in all directions. His breathing is natural, the diaphragm acting normally.

"*Upper limbs.*—There is no muscular wasting. The grasp of the right hand is 35 dynamometer, of the left 20. There is some rigidity about the wrists, especially the left, and the hands have a tendency to drop. During the movements of the arms there is some shaking. The muscular sense is normal.

"*Lower limbs.*—The legs present some 'clasp-knife' rigidity; the thighs resist abduction. His walk is stiff and slow, the left foot apparently dragging more than the right. Tickling the soles of the feet causes excessive reflex.

"The patellar tendon reflex is exaggerated in each knee. There is foot clonus in both extremities, but on the right side more than the left. He is able to walk about a quarter of a mile.

"The functions of the bladder are not impaired.

"There is no loss of cutaneous sensibility anywhere.

"The patient says that he has had no pains at any time in his arms or legs, except a little pain in the stiff shoulder-joints, and this only on movement."

In all probability in this case there was rupture of vertebral ligaments, and possibly also of intervertebral substance, with

hæmorrhage. There may have been displacement also or fracture of a vertebra, but the result leaves this uncertain. It is clear that in some way there was a sudden compression of the cord; and it is probable that, although the source of this compression has gradually been removed, the cord itself has sustained about the point indicated by the patient some permanent damage, which has been followed by secondary degeneration of the lateral columns.

CASE IV.—*Gradually increasing weakness of all four extremities; head rigidly fixed to one side; prominence of third cervical spine; slight paralysis of facial muscles and of palate; absence of movement of diaphragm; respiration, mainly upper thoracic; wide-spread loss of cutaneous sensibility; attack of double pneumonia; imminent peril to life; recovery from pulmonary affection; gradual increase in power of limbs, and recovery of movements of diaphragm.*

On May 14th, 1879, Mary Ann M——, æt. 13, was brought to the National Hospital, Queen Square, with gradually increasing weakness of the four extremities, and was admitted at once as in-patient.

The following notes were taken by Mr. A. E. Broster, Resident Medical Officer:—

"She came from a miserable hovel, where she was living in great poverty, and had been ill-fed. For a few months in the winter she had been in a situation as general servant, where she had to work very hard, and lift heavy weights. It was in December 1878, whilst thus employed, that she complained of sore throat, for which she applied a mustard-plaster, and then bathed her neck in cold water to remove the stinging which this occasioned. Next day she had a stiff neck. Two or three days later, rain came through the ceiling on to her head and body whilst she was asleep in bed. This was followed by pains in the neck and shoulders and the right knee-joint. About the end of January 1879 the right hand is described as having been swollen and blue. The thumb is said to have had little blisters on it. About a week later the right arm got bad, and then her shoulder became powerless. A few days more and the right leg began to fail, and at the end of March

she was forced to leave her place. A fortnight before her admission into the hospital the right arm had got, she says, to its worst, but the leg was still continuing to become more feeble.

"During the last week or ten days the left arm has failed, and the left leg has been getting numb and weak. For three weeks past she has been unable to walk by herself.

"The following was her state on admission:—

"The head is rotated to the right, and immovably fixed in this position, the long cranial axis being at an angle of 45°, with the inter-scapular axis on a plane parallel with and above the latter. The third cervical spine is thought to be somewhat unduly prominent, and slightly displaced to the left. Around and above it there is some hard thickening, apparently beneath the muscles. No tenderness is experienced except at a spot in a line with and below the right mastoid process.

"*Mental condition.*—Normal.

"*Special senses.*—*Smell* more acute on the left than the right side, tested by sumbul and assafœtida.

"*Sight.*—Reads 2½. Snellen left eye, 5½ right eye. Ophthalmoscope shows no change.

"*Hearing.*—Normal.

"*Taste.*—Somewhat deficient on both sides, but especially on the left.

"The upper lip has a somewhat expressionless look. When she smiles, the two angles of the mouth act evenly and fairly, but in voluntary effort, as e.g. in showing her teeth, there is decided weakness on both sides, most marked on the left. She cannot frown. She says she bites her right lower lip. Asked to shut her eyes, the lids fail to meet by ⅛-inch, but by a strong effort and the aid of other facial muscles she can approximate them. The reaction to faradism of the facial muscles is normal.

"The right masseter is thought to be somewhat stronger than the left, the horizontal movement of the lower jaw is good.

"The movements of the eyeballs are perfect. There has been no diplopia. Pupils are equal, moderate in size, and react well to light.

"She speaks clearly, there is slight tremor of the tongue.

Swallowing is imperfect, but liquids have never returned through the nose. The palate goes up more on the left than the right side. Uvula deviates to the left.

"*Tonsils* are large and somewhat reddened. A small ulcer seen on the left.

"*Laryngeal movements* normal. She is unable to move her head in any direction whatever.

"*Trunk.*—Respiratory movements. She says, 'I cannot take my breath so quick as I used to. Sometimes I cannot draw a long breath for two or three minutes, although I feel I want to.' She takes a number of short inspirations.

"When the hand is placed on the epigastric region, and the patient takes as deep an inspiration as possible, the abdominal wall is felt to fall in with the inspiration. There is very little movement of the ribs. There is no incontinence of urine or fæces.

"*Upper extremities.*—The right is very stiff, but no pain is caused by passive extension. There is 'clasp-knife' rigidity at the elbow-joint. She can just slightly flex this joint, and there is a faint power of grasp with the right hand.

"She can place her left hand on the top of her head, but the grasp of this hand is not sufficient to affect the dynamometer.

"The forearms, which are equal in size, measure 7½ inches at their greatest circumference. In the hands there is hyper-extension of the metacarpo-phalangeal joints, owing to weakness of the interossei, especially marked on the right side. There is no diminution of excitability to faradism in the muscles of either arm. From the drooping position of the wrists (especially the right) there is evidently weakness of the extensors.

"*Lower extremities.*—The patient can just stand; she cannot walk. There is marked 'clasp-knife' rigidity in the right much more than in the left. As she lies she can lift the left leg off the bed, not the right, and can draw up both legs, the left most strongly. Calves measure 10½ inches each. There is no foot clonus on either side. The patellar tendon reflex is exaggerated, and is more marked in the right than the left limb.

"*Cutaneous sensibility* is lost in the tips of the right fingers,

front of the right thigh, dorsum of the right foot, and the right shin. It is diminished over the trunk and extremities generally, from the neck downwards behind, and from 2 inches below the level of the clavicles in front, with the following exceptions. Along the ulnar border of the anterior aspect of the right forearm there is scarcely any diminution. Here there is distinct hyperalgesia to the induced current. The left upper arm is nearly normal as to its sensibility.

"There is no alteration of sensibility in the face, nor in the soles of the feet.

"The patient complains of no pain anywhere, but of a sensation of pins and needles in the forearms."

Family history.—Her mother has twice miscarried at the third month. The father has had no signs of syphilis, but had a gonorrhœa in youth. The other children show no signs of congenital syphilis. A brother died, four days before this girl attended the hospital, of rheumatism and bronchitis. Neither he nor any other member of the family suffered from a bad throat. There was no illness at the place where the patient lived as servant.

The patient had scarlet fever in infancy. Never had acute rheumatism, nor glandular swellings, nor injury by accident.

Viscera healthy. Temperature normal. The treatment was rest in bed and iodide of potassium. In the course of four days there was a good deal of improvement, both legs could be lifted off the bed, and there was some increase of power in the arms. The head, too, could be turned a little to the right and left, and bent forward a very little. The breathing remained as before, and Mr. Broster notes, "The pectorals and sterno-mastoids seem to do the work."

A gutta-percha support was fixed to the head and neck.

On June 12th it is noted that "during the last two days the patient has complained of pain in the right leg, shooting down from the groin to the knee, and there has been more difficulty in swallowing. There is wasting of the interossei."

Next day there was orthopnœa, the respirations very rapid, and entirely upper thoracic. There was some cyanosis. These symptoms increasing on the following day, the headgear was removed, as it was thought it might interfere with

the action of the sterno-mastoids. Auscultation showed tubular breathing and crepitant râles along the base of the left lung, with harsh respiration in the right lung. There was very slight cough. The alæ nasi flapped. Her temperature was 101·4.

On the evening of the 15th her temperature was 103, the pulse 130, dicrotous. Respirations 36. She was very cyanotic. There were crepitant rhonchi throughout the left lung behind, and along the axillary border of the right. There was some delirium. Deglutition was much impaired, so that she could only swallow very small quantities of fluid. With great difficulty and gasping she stated that there was some pain in her head. At this time death appeared inevitable. After this there was gradual improvement in the respiratory system, but the loss of power in the extremities was at first more pronounced than it had been before the commencement of the pneumonic attack.

On the 18th I observed that the left side of the chest moved a little more than the right; the movement being mainly an elevation of the upper ribs; there was but very little lateral expansion. The epigastrium sank during inspiration.

On June 27th it is noted that the temperature is 100°; the respiration mainly upper thoracic, but with a little unilateral expansion. Over the base of the left lung the percussion note was dull, and there was increased vocal fremitus. Moist bubbling and crackling râles could be heard. On the right side there were a few small mucous râles along the posterior border.

Both legs could now be moved freely; each could be lifted off the bed and held extended for about thirty seconds.

By the middle of July all traces of the pneumonic attack had subsided. There was gradual improvement in the paralysed limbs. In the middle of September she had so far recovered that she was able to be sent to the Convalescent Establishment at Finchley, and the following note of her condition was then taken:—"She carries her head in the same 'side way' as on admission, and has gained no power as yet over its movements. She cannot look over her shoulder. The thickening previously described is very distinct around and over the third cervical spine. This spine can be felt distinctly prominent

beyond the other cervical spines. The weakness described in the facial muscles remains *in statu quo,* except that she can approximate her eyelids a little more than she could formerly. There is some deviation of the uvula to the left. The tonsils are large. The respiration is entirely thoracic, no movement of the diaphragm being perceptible.

"*Upper limbs.—Right.*—She can flex her elbow; the limb is slightly stiff. There is a perceptible, but not measurable, grasp of the hand. No distinct wasting of muscles. She can raise the arm to the level of the shoulder.

"*Left.*—She can hold up the arm fairly. The grasp of hand is not measurable. She can feed herself, and cut up meat, but cannot dress herself.

"*Lower limbs.*—She can stand and walk; the calves each measure ten inches in circumference. The walking is performed in a shaky manner, the trunk held stiffly, and the knees bent. She does not drag her feet. In rising from a chair she appears to have much difficulty, and requires a little impetus before she can get on to her legs.

"The patellar tendon reflex is exaggerated on each side. Foot-clonus is present, though not to a very marked extent, in both limbs—in the right more than in the left.

"*Cutaneous sensibility.*—On the left hand and forearm (except in the tips of the fingers, which are still numb) sensation is normal. On the left leg it is deficient.

"*Right upper limb.*—Sensibility is still deficient, though less so than formerly. On the right leg it is as good as on the face, where it is apparently unimpaired.

"For her own part, the patient says that the sensation in the left lower limb is better than it was; and that in the right leg, which used to be the worst, she feels better than in the left. The right leg, which was the weaker, is now, she says, the stronger. She says that a touch upon her back is felt quite right, but not so on the front of the body. She has no pains whatever, and no sensations of 'pins and needles.'

"Appetite good. She has control over the sphincters. Two months and a half later, whilst at Finchley, she was well able to get about by herself. The movement of the diaphragm had returned."

My first impression, on seeing this patient in the out-patient room, was that we had to do with one of those acute cases of nearly universal paralysis, the pathology of which remains obscure, and of which I have brought two marked examples before the Clinical Society of London. One had to consider the possibility also, in view of the history of "sore throat," that the affection might be diphtherial. Both these contingencies were disposed of by the observation of the fixed position of the head, and the discovery of the thickening about the third cervical spine. The case resolves itself, therefore, in all probability, into one of Pott's disease, high up in the cervical region. If this be so, the internal lesion (pachymeningitis), besides cutting off nervous impulses from the extremities, ribs, and diaphragm, apparently involves also to a certain extent the origins of the facial and glossopharyngeal nerves.

The pains were not strongly marked. Such as were present affected the head, doubtless through the occipital nerves.

The most interesting point in this remarkable case is the long-continued paralysis of the diaphragm. This paralysis was doubtless only partial, like the paralysis of the extremities and of the intercostals; but it was pronounced enough to give rise to the abdominal depression during inspiration mentioned by Duchenne as characteristic of paralysis of this muscle. Duchenne thus describes the condition. "At the moment of inspiration the epigastrium and the hypochondria are depressed, whilst, on the other hand, the chest dilates; the movements of these parts are reversed during expiration." Recovery from such an attack of pulmonary inflammation as this girl suffered must, in the circumstances of her case, be an exceedingly rare event.

DISTRIBUTION OF THE ARTERIES OF THE SPINAL CORD.

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A comparison of fortunate sections of the spinal cord has enabled me to map out pretty accurately the distribution of its vessels. The *anterior median*, the *posterior spinal*, and the *central arteries* are already well described in ordinary anatomical works, and need only be mentioned here.

The *anterior median artery* gives a series of small branches, which pass backwards in the anterior median fissure, to reach the anterior commissure; hence these vessels may be called the arteries of the anterior median fissure (Fig. 1, *a f*). Each of these vessels on reaching the anterior commissure divides into two main trunks, each of which enters the grey substance of the anterior horns, and which may be called the arteries of the anterior commissure (Fig. 1, *a c*). Each trunk subdivides into three branches, which, from their position, may respectively be named the anterior (Fig. 1, 1), median (Fig. 1, 1′), and posterior (Fig. 1, 1″) branches of the artery of the anterior commissure. The anterior branch curves forwards, and is distributed to the anterior and internal portion of the grey substance; the median is distributed to the lateral portion of the anterior horn, while the posterior is directed backwards to the posterior horn. The central artery also gives off an anterior (Fig. 1, 2) median (Fig. 1, 2′) and posterior (Fig. 1, 2″) branch, which are distributed respectively to the anterior, lateral and posterior portions of the grey substance. The median branches of the two main vessels, besides supplying the grey substance, are also distributed to

the pyramidal tract of the lateral column. The posterior spinal artery (Fig. 1, *p a*) gives off branches, which pass by the

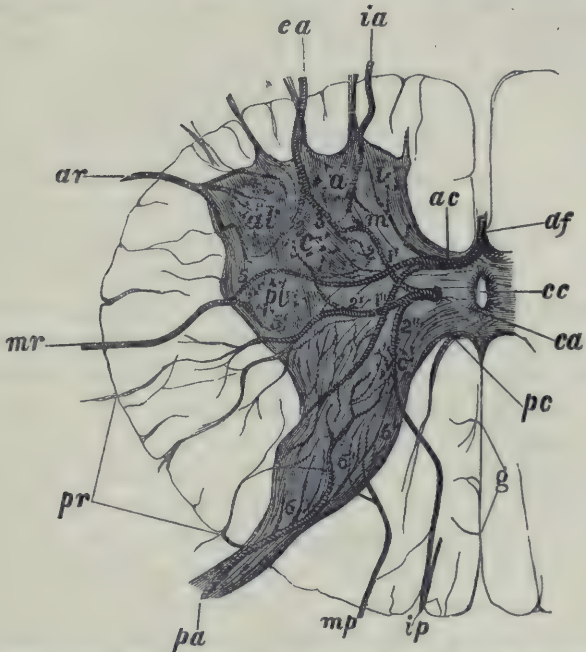


FIG. 1.—SCHEMATIC REPRESENTATION OF THE DISTRIBUTION OF THE BLOOD-VESSELS IN THE CORD.*

Anterior median artery.
a f Arteries of the anterior median fissure.
a c Artery of the anterior commissure.
 1 Anterior branch.
 1′ Median branch.
 1″ Posterior branch.
c a Central artery.
 2 Anterior branch.
 2′ Median branch.
 2″ Posterior branch.
p a Posterior root arteries.
6 6′ 6″ Arteries of posterior horns.
i a Internal anterior root artery.
e a External anterior root artery.
 3 3′ Internal and external branch.
a r Antero-lateral branches.
 4 Anterior branch.
m r Median lateral artery.
 5 5′ Anterior and posterior branches.
p r Posterior lateral arteries.
i p Internal posterior artery.
m p External posterior artery.
g Arteries of the column of Gull.
p c Artery of the posterior commissure.
v c Vascular column of Clarke.
i Internal group of cells.
a Anterior group.
a l Antero-lateral group.
p l Postero-lateral group.
c Central group.
m Median group.

side of the posterior roots to enter the grey substance of the posterior horns, where they subdivide into a variable number of small branches (Fig. 1, 6 6′ 6″), which may be called arteries

* I have to thank Dr. Young, of the Owens College, for the above diagram.

of the posterior horns. In addition to the vessels just described, a large number pass from the pia mater into the substance of the cord, and some of these are so large and so constant as to deserve special mention. Two of these run by the side of the bundles of fibres which constitute the anterior roots of the nerves, hence they may be called the anterior-root arteries. The branch nearest the median fissure may be called the internal anterior root (Fig. 1, *i a*), and the other the external anterior-root (Fig. 1, *ea*) artery. The internal artery on entering the grey substance joins the anterior branches of the first subdivision of the artery of the anterior median fissure and of the central artery. The external anterior-root artery on entering the grey substance subdivides into two branches, the inner (Fig. 1, 3) of which is distributed along with the vessels just mentioned, while the outer branch (Fig. 1, 3′) passes between what we may call the antero-lateral (Fig. 1, *a l*) and central groups (Fig. 1, *c*) of cells. A very constant vessel passes to the grey substance from the pia mater, at the point of junction of the anterior and lateral columns of the cord, and it may therefore be called the antero-lateral artery (Fig. 1, *a r*). On reaching the grey substance it frequently divides into three branches, one of which passes in front (Fig. 1, 4), another behind, and another into the substance of the antero-lateral group of cells. Another constant vessel (Fig. 1, *m r*) passes from the lateral aspect of the cord, and on reaching the grey substance it subdivides into two branches, the one of which passes in front and the other behind the postero-lateral group of cells (Fig. 1, *p l*), and this vessel may from its position be called the median-lateral artery. Small branches (Fig. 1, *p r*) pass at short intervals from one another through the posterior part of the lateral column, and, together with the median branches of the first subdivision of the artery of the anterior median fissure, and of the central arteries, supply the posterior part of the lateral columns; hence these vessels may be called posterior-lateral arteries. Two vessels pass from the pia mater into the substance of the posterior column, the one nearest the posterior median fissure, and which may therefore be called the internal posterior artery (Fig. 1, *i p*), passes between the column

of Goll and the posterior root zone ; and after passing through about two-thirds of the depth of the posterior column, it curves outwards to reach the posterior grey horn. The other vessel may be named the external or median posterior artery (Fig. 1, *m p*) ; it passes into the substance of the posterior column at the middle of the posterior root zone, and on reaching about one-third the depth of the posterior column, it also curves outwards to reach the posterior grey horn, where it terminates. Small vessels (Fig. 1, *g*) pass from the pia mater, in the posterior-median fissure, into the substance of the column of Goll.

Another vessel, which may be called the artery of the posterior commissure (Fig. 1, *p c*), passes from the pia mater along the posterior margin of the posterior commissure, and winds backwards along the internal edge of the posterior horn.

With regard to the groups of cells in the anterior horns, I have, on the whole, followed the classification of Erb. These groups are the vesicular column of Clarke (*v c*), the postero-lateral (*p l*), and the antero-lateral groups (*a l*). The group which Erb calls anterior, I propose to call internal (*i*), because a very constant group of caudate cells is always observed near the anterior roots, which from its position is best distinguished as the anterior group (*a*). Also the group which Erb calls the median, I propose to call the central (*c*), because I wish to distinguish a very important area (*m*), which has hitherto not been described, and which from its position and connections is best denominated the median area. The cells of the median area are caudate, but they are much smaller than those of the other groups. This area is only present in the cervical and lumbar enlargements, the portions of the cord which regulate the movements of the limbs; its relatively larger size differentiates the anterior grey horn of the cervical enlargement of the adult human cord from the lumbar enlargement; in the human embryo at the fifth month and in the cord of the ox and dog, this area is of the same relative size in both the cervical and lumbar enlargements, and the anterior horns are under such circumstances almost indistinguishable; hence it may be presumed that in the median area are mainly organised the complicated movements which distinguish the hand of man from the anterior extremity

of the lower animals, so far as these movements are represented in the spinal cord. Other interesting facts tending to the same conclusion are that the cells of the median area in the human cord only assume processes after birth; while those of the other groups possess distinct processes at the fifth month of embryonic life.

The median area is entirely absent in the upper cervical and dorsal portions of the cord, and consequently the internal, anterior, antero-lateral, and central groups approximate, so that they are not always readily distinguishable. In these regions, however, an area is interposed in the human cord between the antero-lateral and postero-lateral groups, which possess some of the characteristics of the median area in the cervical region. The cells of this area are relatively small, they are comparatively late in development, and do not possess distinct processes at birth; and the area is entirely unrepresented in the cord of the ox and dog; hence it may be inferred that this area represents the additional organisation rendered necessary by the maintenance of the erect posture in man.

It may be readily imagined that in all inflammatory diseases the tissues in the vicinity of the vessels will be more liable than the remote portions to be inundated by effusion; hence the lines of the distribution of the vessels may be said to form lines of least resistance to disease. It is also worthy of remark that the development of the cells proceeds from the centres of the groups to their margins, and that in progressive degenerative diseases, the degeneration proceeds from the margins of the groups to their centres. To this law the median area in the lumbar and more especially in the cervical enlargement and that which lies between the antero-lateral and postero-lateral groups in the dorsal and upper cervical regions, are apparent exceptions. These areas, although containing cells, can hardly be called groups; they are probably areas where the marginal cell of the real groups meet. But whatever may be their nature they are the most vulnerable portions of the cord, and as they are the last to be developed, so they manifest the greatest liability to disease.

Critical Digests and Notices of Books.

Nothnagel on the Regional Diagnosis of Diseases of the Brain. (Topische Diagnostik der Gehirnkrankheiten. Eine Klinische Studie. Von Dr. Hermann Nothnagel. Berlin, 1879. Hirschwald, 8vo. pp. 626.)

THIS is an exceedingly able and important work, and worthy of the author's reputation as a physiologist and physician. It is an elaborate collection and digest of the more important facts and observations relating to the regional diagnosis of diseases of the encephalic centres, from the medulla oblongata up to the cerebral cortex and its individual parts. After recording and analysing the clinical facts bearing on disease of each particular region, the author sums up the conclusions which he considers they justify in a series of lucid diagnostic propositions.

Though naturally the work is largely of the nature of a compilation, yet the author displays much originality and critical acumen, and the whole bears impress of a mind thoroughly capable of dealing with every detail. It professes to deal only with the clinical aspect of the question. Physiological and anatomical considerations are reserved for treatment in a separate work. This will no doubt be looked for with interest by all who believe that anatomy, experimental physiology, and classical medicine ought mutually to support and shed light on each other. As to the propriety or even possibility of entirely abstracting from anatomical and physiological researches in a work devoted to the regional diagnosis of cerebral lesions, and therefore by implication determining the functions of the parts involved, there may be some difference of opinion. It may be possible, and perhaps advisable, to avoid entering

at length into these topics, but to exclude them entirely is not capable of being carried out; and in truth it may be asserted that but for the light of physiological research, clinical facts relating to cerebral disease would still be largely in a state of chaos. And certainly if the clinical evidence here furnished were all the basis on which the diagnostic propositions are based, we should have to regard the foundation as in many respects of an exceedingly slender character. Throughout the whole of this work, in fact, the experimental physiologist is clearly visible, and not unfrequently we find a tendency to draw conclusions, evidently in favour of some theory otherwise arrived at, from clinical facts which are neither sufficiently numerous nor of such a nature as to justify any conclusion whatever. That which renders analysis and conclusions from clinical facts alone extremely difficult and even hazardous, is the extreme complexity of the conditions, and the impossibility of distinguishing in all cases between direct and indirect effects of lesions. Though Nothnagel adopts Charcot's rules as to the necessity of excluding all cases where the lesions are of such a nature as obviously to produce general perturbation and far-reaching disturbance, and to consider as bearing on regional localisation only such lesions as are not likely to cause such indirect effects; yet this from a purely clinical point of view is very difficult to carry out. Brown-Séquard's reading of clinical facts has led, as is well known, to conclusions very different from those usually accepted and here advocated. And we see in our author but too frequently an assumption of indirect action where the clinical facts are at variance with his own hypothesis, and ignoring it when it might be highly important to take it into consideration. That the conclusions of Nothnagel will be more readily accepted than those of Brown-Séquard will depend not merely on the clinical evidence here adduced, but on evidence otherwise obtained, and under the circumstances indispensable.

Our author, however, occasionally forgets another very important principle, though he does acknowledge its validity, viz. to found no conclusions as to the effects of certain lesions unless these effects are constant or their absence satisfactorily accounted for. Negative instances, cases of lesions without

such symptoms, are sufficient to overturn a whole host of positive instances, where the facts seem to establish a causal relation between the lesion and the symptoms, while in reality the symptoms are dependent on some other condition. The history of the localisation of cerebral disease shows how precarious conclusions from clinical evidence alone have been, the conditions of strictly scientific evidence not having been fulfilled.

The manner in which our author in his first chapter treats of diseases of the *Cerebellum* strongly suggests the idea that his chief object is to prove by clinical facts that it is only the middle lobe that is concerned in the co-ordination of equilibrium, &c., and that the disorders characteristic of cerebellar disease only occur where the middle lobe is implicated.

The characteristic symptoms of cerebellar disease are a reeling and staggering gait and vertigo. These, however, are not absolutely pathognomonic of cerebellar disease, as they may occur from other affections of the central nervous system. The diagnosis must take other negative and positive phenomena into consideration. As accessory symptoms, but not due to the cerebellar lesion as such, we have the general phenomena indicative merely of intracranial disease, particularly tumours, viz. headache, vomiting, optic neuritis; and others produced indirectly, such as affection of speech, affections of cranial nerves, and general motor disturbances paralytic or spastic.

The special symptoms are in all cases referred to implication of the middle lobe, either directly or indirectly.

What the standard is by which the author discriminates between direct and indirect action is very difficult to discover from a study of the facts here presented, as well as from others which might be referred to. It has the appearance of being extremely arbitrary and dogmatic. It may be admitted, and clinical facts certainly favour the view, that lesions of the middle lobe are more frequently associated with cerebellar symptoms than when the lesion is in the lateral lobe, but this is very far from carrying with it the conclusion here maintained, that the middle lobe alone exercises those functions generally looked upon as cerebellar. Another explanation and one more in accordance with physiological facts, which,

though not mentioned, cannot be ignored, is that a lesion of the middle lobe is more likely to derange the cerebellum as a whole by affecting all its lobes at once. Our author even thinks that the facts—more particularly those relating to atrophy of the cerebellum—warrant him in advancing the hypothesis that the lateral lobes are "somehow related to the psychical functions." This hypothesis is, it is true, stated with some reserve; but, considering the kind of evidence offered, it would have been better perhaps to reserve it altogether.

Though several of the accessory phenomena, obviously dependent on indirect action on other structures, such as motor paralysis, spasms, &c., are mentioned as occurring in connection with cerebellar lesions, it seems somewhat strange that no particular notice is taken of the rigidity and tetanic-like seizures described so carefully by Hughlings-Jackson in connection with cerebellar tumours, so characteristic as to be almost pathognomonic. Even though indirect, these phenomena are of exceeding value in a diagnostic point of view, and too important to be dismissed among minor accessory symptoms.

The chapter on the cerebellum is certainly not the most satisfactory in the book.

As regards lesions of the *crura cerebelli*, the clinical facts do not allow us to decide positively as to the effects of lesions of the anterior or posterior peduncles respectively. Conclusions as to the special symptoms of lesions of the middle peduncles are also doubtful, owing to the fact that lesions are rarely accurately circumscribed. The most marked phenomena met with are distortion of the axes of the trunk, head, and eyes—particularly the latter—with vertigo and tendency to fall to one side. These our author looks upon as purely irritative phenomena, as in one case at least (Nonat's) the symptoms were not permanent, though the lesions remained. The rotation may be towards, or away from, the seat of lesion. This is not explained, though a fact of this kind might suggest doubt as to whether the symptoms can in all cases be regarded as purely irritative. Destructive and irritative lesions would be the converse of each other, and this may be the real ground of the

difference as to the side towards which rotation occurs. This, however, the author does not take into consideration.

Affections of the *Pons Varolii* are treated separately from those in which the medulla oblongata is also involved, though the two centres are very commonly implicated together.

Hæmorrhages in the pons are rare as compared with those in the hemispheres. Can we diagnose with certainty a hæmorrhage into the pons? Not always, says our author. The chief indication is the fact of alternate paralysis. Thus an apoplectic attack, with alternate paralysis of the limbs and face, with anarthria and dysphagia, points to the pons as the seat of lesion. Of great importance practically are signs of motor irritation, in the form of general epileptiform convulsions, considered pathognomonic by Ollivier. These convulsions Nothnagel explains by irritation of his so-called "convulsion centre," though why such a term should be employed, and an animal credited with a centre for the display only of pathological effects, it is not easy to comprehend without an abuse of physiological terms. That convulsions should readily occur from irritation of the pons, where all the sensory and motor strands of the encephalon are in such close relation, is a fact not difficult to understand without the questionable aid of such an objectionable term. These convulsions, however, are not constant, and Nothnagel does not agree with Ollivier in regarding them as of themselves pathognomonic. Vomiting is common, but not invariable. The state of the pupils in pontine hæmorrhage is one to which great importance has been attached. Very often they are contracted. Hence this condition, combined with complete muscular relaxation, simulates narcotic poisoning. The pupils, however, have been found dilated, or of normal size, and the reaction to light may also vary. Sudden death—fulminating apoplexy—is not uncommon from hæmorrhage into the pons, probably owing to indirect action on the medulla oblongata.

Neither the occurrence of albuminuria, nor of diabetes mellitus, though sometimes met with, can be taken as constant in pontine lesions. Similarly, also, as regards affections of circulation and respiration. These latter are to be ascribed to action on the medulla oblongata.

Prevost's idea that in pontine hæmorrhage the conjugate deviation of the head and eyes is away from the side of lesion instead of towards it, as in cerebral hæmorrhage, is not substantiated.

As a general result it is stated that a hæmorrhage into the pons can only be diagnosed with certainty "when there is alternate paralysis of the face and extremities. It may be considered probable when the apoplectic attack is accompanied by irritative motor-phenomena in the form of general convulsions, with contracted pupils, and when death occurs within a few hours. Under all other circumstances only conjectures are possible" (p. 105).

Embolism of the basilar artery is stated by Nothnagel not to occur. This, however, has been controverted by Gowers in a recent communication to this Journal. ('BRAIN,' No. VIII. "Illustrations of Diseases of the Pons Varolii.") Thrombosis is not uncommon. This may be rapidly fatal or lead to chronic softening, with "herd-symptome." These symptoms, which occur with hæmorrhagic or necrotic softenings, are very numerous.

Motor paralysis of the limbs on the side opposite the lesion may be seen—more marked in the lower than upper extremity—without affection of cranial nerves; or the cranial nerves may alone be paralysed; but more commonly there is alternate paralysis of the extremities on the opposite, and of cranial nerves on the same side as the lesion. But the face and limbs may be paralysed on the same side. This occurs when the lesion is nearer the crus cerebri; the alternate form occurring when the lesion is nearer the medulla. The facial paralysis in pontine lesions resembles peripheral facial paralysis, both as regards the diminution of faradic excitability, and as regards the reaction of degeneration to galvanism.

Besides the facial we may have affection of the trigeminus, abducens, (auditory?) and hypoglossal. The hypoglossal is affected next in frequency to the facial, and then the abducens. The affection may be on the side of lesion or on the opposite. Whether a conjugate ocular paralysis of the rectus externus on the same side, and rectus internus of the opposite side is characteristic of pontine lesion, is uncertain.

Double motor paralysis of the limbs is not common. Contractures in paralysed limbs often occur with stationary lesions, but not epileptiform convulsions. Occasionally ataxic symptoms have been observed.

Affections of sensibility also occur, either in the form of anæsthesia or hyperæsthesia in the paralysed parts. There may be facial anæsthesia on one side and anæsthesia of the limbs on the opposite.

The symptoms produced by tumours are very variable and difficult to distinguish from affections of the *basis cranii*. Some tumours remain latent; others cause alternate paralyses, or affections of cranial nerves, or disturbances of equilibrium.

The various facts here indicated are generalised in the diagnostic propositions at the end of the chapter, which is one deserving the highest praise, and which with its elaborate *résumé* and digest of cases will be of signal use to all neurologists.

The diagnosis of affections of the *crura cerebri* rests on the occurrence of paralysis of the oculo-motor on the side of lesion, and of the extremities on the opposite—the best marked instance of which is Weber's well-known and much-quoted case. Such a form of alternate paralysis can only occur with a lesion at the base of the brain. The situation in the crus is to be determined mainly by the mode of onset. If it occur suddenly, the crus is indicated—not otherwise.

Occasionally from tumours both third nerves may be implicated.

Lesions confined to the *corpora quadrigemina* in the form of hæmorrhage and softening do not exist. Hence conclusions as to the special effects of lesion of the corpora quadrigemina have to be founded almost entirely on tumours, affecting these ganglia in whole or in part, and for this reason very uncertain. All the published cases are considered in detail, and the following conclusions are based on them. Lesions of the anterior tubercles are almost always, if not always, associated with blindness. We may perhaps diagnose a lesion here, if we have along with the general indications of a cerebral affection sudden amaurosis with immobility of the pupils, without any abnormal ophthalmoscopic appearances. In cases of lesion of

the posterior pair we may have affection of the oculo-motor nerves. This, however, is dubious. The character of the affection is peculiar. Thus a bilateral affection of symmetrically functioning branches, such as double ptosis or double impairment of certain ocular movements, points to the corpora quadrigemina, especially if no alternate paralysis of the extremities exists. This bilateral affection may result from unilateral lesion. In connection also with lesions of the posterior tubercles, disorders of co-ordination, equilibration, &c., have been observed, similar to those of cerebellar lesion. This subject, however, requires further investigation.

Lesions of the *optic thalami* are treated at considerable length, as the obscurities and uncertainties surrounding the question require. According to the general plan followed, recent hæmorrhages and softenings, stationary lesions and tumours are successively discussed.

Hæmorrhages confined to the optic thalami, and not also implicating the corpora striata, are much less frequent than generally stated to be the case. There are no symptoms which enable us to diagnose with certainty a hæmorrhage into the optic thalami as such, at least at the beginning. The occurrence of tonic or clonic spastic phenomena, on which great reliance is placed by many observers, is not constant, nor are they pathognomonic of lesions of the optic thalami, as they occur also with hæmorrhages in the centrum ovale, cortex, pons, and crus cerebri. The symptoms met with in connection with chronic lesions are grouped under different heads, and illustrated by reported cases. (1) Some lesions, single or double, may be entirely latent; (2) in others then we have anæsthesia of the opposite side, without motor paralysis; (3) in a third group we have hemiplegia without affection of sensibility; (4) in a fourth we have hemichorea or athetosis, without loss of sensibility or motor paralysis; and in a fifth we have affections of vision. The symptoms, therefore, are exceedingly complex and contradictory, and the question is in how far the optic thalamus as such is related to the symptoms described.

It used to be taught by Saucerotte, and more recently advocated again by Lussana and Lemoigne, that the optic thalamus was more especially related to the upper extremity.

This, however, Nothnagel repudiates. He reiterates his view formerly expressed, that lesions accurately limited to the optic thalamus occasion no motor paralysis whatever.

Motor paralysis, if it does occur in connection with lesion of the optic thalamus, occurs most frequently when the lesion is in the middle third, and is explicable by implication of the internal capsule. Athetosis and post-hemiplegic chorea are, as is well known, ascribed by Charcot to lesion of the posterior part of the internal capsule, but Nothnagel is not satisfied that the optic thalamus is not also concerned in the causation. As to affections of cutaneous sensibility, he does not think they are related to lesion of the optic thalamus as such, but the question is not altogether definitively settled.

With respect to Crichton-Browne's view that lesions of the optic thalami are associated with abolition of reflex excitability, our author does not think it has been conclusively established, but he is disposed to accept it as probable.

The relation of the optic thalami to "psycho-reflex" movements is considered. In those cases where there is paralysis of volitional facial movements while the emotional continue, Nothnagel thinks the optic thalami and their cortical connections must be intact; while paralysis of the emotional with retention of the volitional in all probability will indicate lesion of these parts. This question, however, is one requiring further investigation. As to the relation of the optic thalamus, if any, to the muscular sense, we know nothing as yet at all definite.

The question as to the relation of the optic thalamus to vision depends mainly on two reported cases, Remy's and Hughlings-Jackson's. In the former it is doubtful whether there was crossed amblyopia or bilateral hemiopia; in the latter hemiopia was distinctly made out. Nothnagel, however, expresses considerable doubt as to whether the hemiopia can be regarded as dependent on the optic thalamus lesion, inasmuch as the same may result from lesion of the optic tract, and also from lesion of the occipital lobe—a question discussed below.

Vaso-motor disturbances are not specially related to optic thalamus lesions.

Our author would—but with reserve—in diagnosing a lesion of the optic thalamus lay special stress on affection of vision in connection with lesion of the posterior part, on the occurrence of hemichorea, athetosis or tremor, abolition of reflex excitability, and possibly of the muscular sense.

Lesions of the *corpora striata* are also discussed very fully, and on a consideration of the clinical facts he comes to the following conclusions.

Chronic lesions of the corpus striatum may be accompanied by motor, sensory and motor, and vaso-motor paralysis on the opposite side. Unless the lesion is very small the motor paralysis is a constant symptom. The hemiplegia may disappear if only the nucleus-caudatus or nucleus-lenticularis is affected. In cases of persistent paralysis—which occurs when the internal capsule is injured—secondary contracture frequently sets in. The motor paralysis usually affects both extremities, the lower facial region, and also to some extent the thoracic and trunk muscles. The hypoglossus is either not affected, or only temporarily, very seldom permanently. In rare cases only the extremities, or the facial movements alone are affected. It is impossible to distinguish between lesion of the nucleus caudatus and nucleus lenticularis. Motor paralysis is the only symptom where the lesion is confined to the anterior part of the corpus striatum, or the region supplied by the lenticulo-striate arteries. In some cases hemianæsthesia accompanies the hemiplegia. This hemianæsthesia is characterised by the implication of the special senses along with cutaneous sensibility on the side opposite the lesion; but the hemianæsthesia may be confined to cutaneous sensibility alone. Usually hemianæsthesia and hemiplegia co-exist. Only exceptionally does the latter disappear, leaving the anæsthesia. The existence of hemianæsthesia points to lesion of the posterior part of the internal capsule. The lesion is also frequently accompanied by vaso-motor disturbances on the paralysed side.

Hemichorea often occurs along with anæsthesia, but its relation if any to the corpus striatum is doubtful.

Centrum Ovale.—In considering and localising lesions of the centrum ovale Nothnagel arrives at essentially the same conclusions as Pitres (Lésions du Centre Ovale). He, however,

suggests some modifications of his "sections" and nomenclature—not, however, of much importance.

Lesions are latent when they occupy the occipital, sphenoidal, and frontal (anterior and middle) sections. Tumours in these regions may also remain latent as regards special symptoms, only those characteristic of intracranial tumours in general existing.

Even if special symptoms exist in connection with lesions of the centrum ovale, it is not possible to fix the seat of lesion with certainty, as the same symptoms may occur from cortical lesions, or even from lesions of the corpus striatum. The special symptoms, which occur only with lesions in the fronto-parietal region, are either general hemiplegia, or monoplegia, as in cortical lesions. Our author considers it doubtful if convulsive spasms occur when the lesion is strictly confined to the centrum ovale, and does not implicate the cortex; but early rigidity may occur, as argued by Pitres, from irritation of the cortico-striate medullary fibres. We have no sufficient material for forming any conclusions as to the relation of lesions of the centrum ovale to trophic or vaso-motor disturbances. Aphasia may occur in connection with lesions in the medullary fasciculi of the third left frontal convolution.

Cortex Cerebri.—In treating of lesions of the cortex our author again expressly states that he founds his conclusions on clinical facts alone—entirely abstracting from physiological considerations. His conclusions as regards affections of motility agree in all essential respects with those of Charcot and other writers on cerebral localisation. That is, that in lesions strictly cortical affecting the anterior and posterior central convolutions, and paracentral lobule, we have either hemiplegia, or monoplegia, according to the position and extent of the lesion in these regions. As regards the exact locality of the lesions causing the different forms of monoplegia, there is little or nothing requiring special mention in connection with the literature of this subject.

The cortical position of the lesion is to be diagnosed not so much from the paralysis itself but from the accessory phenomena, such as local or generalised irritative phenomena, &c.

Hemiplegia with aphasia points to cortical origin; also hemiplegia associated with ptosis.

Nothnagel, however, does not accept the view advocated by Landouzy that ptosis indicates lesion of the parietal lobule (angular gyrus). In reviewing the cases which have been brought forward as destructive lesions of the motor zones without paralysis, he holds that there is not a single unequivocal case on record. Either the position of the lesion is not accurately given, or the nature of the lesion is such as not necessarily to destroy or entirely abolish functional activity. He justly ridicules a collection of cases by Lussana and Lemoigne as of no scientific value whatever.

Before passing from Nothnagel's consideration of the motor affections in connection with cortical lesions, we may observe that paralysis of the tongue from cerebral lesions is spoken of as paralysis of the hypoglossus, and similarly in regard to other monoplegiæ. By the term paralysis of the hypoglossus a peripheral paralysis is apt to be suggested, though not intended. It would be well to avoid terms which are likely to create confusion and misconception.

The affections of sensibility which our author attempts to connect with lesions in certain parts of the cortex are affections of the muscular sense, and affections of sight—particularly hemiopia. The evidence adduced on these heads, however, is not of a very satisfactory character.

He admits that affections of cutaneous sensibility have no relation to cortical lesions, and yet on the strength of two or three cases, complicated either with cutaneous paræsthesia or anæsthesia, and not one of which can be regarded as admissible in evidence, the lesions being either multiple, or extending deeply into the brain-substance, he ventures to conclude that probably affections of muscular sense are due to lesion in the parietal region of the cortex, a region near, but not identical with, the motor regions—a region, likewise, in which lesions are frequently if not generally found to be latent.

In formulating such conclusions our author seems to violate the principles by which he professes to be guided in reading clinical facts.

The very cases here related may be interpreted much more in accordance with established clinical facts, by lesion direct or indirect of the posterior part of the internal capsule and its neighbourhood.

Our author refers to two distinct affections of sight in connection with cortical lesions, (1) double hemiopia, (2) crossed amblyopia.

He attempts to bring hemiopia in relation with lesion of the occipital lobe—the retina being affected on the side of the lesion. That lesions of the occipital lobes may be altogether latent is also admitted.

The cases on which the relation between hemiopia and lesion of the occipital lobe is founded are far from satisfactory. In Pooley's case there was also a lesion in the optic thalamus; in Hirschberg's there was a tumour reaching to the optic thalamus; Wernicke's was a case of multiple lesion, both cortical and medullary; in Baumgarten's there was also lesion of the optic thalamus; and Nothnagel's own case was one of diffuse and multiple lesion in both hemispheres.

On material such as this it is surely hazardous to found conclusions.

Moreover, when we consider that it has been clearly shown that hemiopia occurs from lesion of the optic tract, and from lesion of the optic thalamus posteriorly, we require very stringent evidence that these parts were free from implication, directly or indirectly, before we can admit any causal relation between occipital lesion and hemiopia as being made out. In a case recently brought before the Soc. Méd. of Geneva by J. L. Prevost, in which hemiopia was very definitely determined, there was extensive occipital lesion, but also lesion of the posterior part of the optic thalamus and external geniculate body.

Though Nothnagel seems to be very doubtful of his own conclusions in his analysis of the facts, he is not so careful elsewhere, and one might be led to consider the proposition established, which at present at least it is not. While he with some degree of confidence connects hemiopia with lesions of the occipital lobe, he does not offer any suggestion as to the locality of the lesion which produces crossed blindness. This

symptom has been chiefly observed by Fürstner in general paralytics.

Nothnagel thinks that the connection between "word-deafness" and lesion of the superior temporo-sphenoidal convolution fairly well-established. As regards aphasia, his conclusions are essentially those of Kussmaul. We have no definite clinical facts enabling us to connect affections of smell and taste with cortical lesions. Similarly as regards trophic and vaso-motor disturbances. Psychical disturbances are as yet beyond localisation.

As to the effects of lesions limited to the *cornu ammonis* we at present know nothing. The reported facts as to the occurrence of sclerosis and atrophy of the hippocampus in epileptics are briefly reviewed, but no causal relationship is considered to be made out. There are likewise no symptoms enabling us to diagnose lesion in the *claustrum* or *external capsule.*

As to the *ventricles,* there are no definite symptoms indicating effusion in one or other as distinct from effusions elsewhere. A diagnosis of lesion of the fourth ventricle may be made if after an injury to the head there should appear diabetes mellitus or insipidus, with general cerebral symptoms such as headache, vertigo, vomiting, and possibly also slowing of the pulse and mental hebetude. The diagnosis will of course be rendered more precise by the occurrence of symptoms special to lesions of the pons and medulla already described.

Affections of the *basis cranii* are very variable and complex. Apart from general indications of intracranial disease, the diagnosis must be founded chiefly on the implication—destructive or irritative—of the cranial nerves, unilaterally or bilaterally, according to the position of the lesion in the anterior, middle, or posterior fossa; together with indications of affection of the cerebro-spinal tracts.

Lesions confined to the anterior fossa implicate the olfactory, and also the optic if they extend backwards.

In the middle fossa the optic, third, fourth, and sixth cranial nerves are specially liable, and also the olfactory if the lesion extends forwards. The facial can only be affected if the lesion extends into the hiatus of the aqueduct of Fallopius.

Lesions in the posterior fossa may affect the fourth, sixth,

and seventh (auditory and facial), the eighth and ninth. The third may also be implicated if the lesion extends forwards towards the middle fossa. With lesions of the posterior fossa also disorders of co-ordination specially occur.

Lesions of the *pituitary gland* cannot be distinguished from other affections of the middle fossa. Nor can *aneurism of the basilar artery* be made out with certainty or distinguished from lesions of the pons from other causes.

At the end of the work there is an "Ueberblick über die Herdsymptome," which is a kind of *catalogue raisonné* of the various facts detailed in the body of the work. In this there is a tendency to speak somewhat more positively than the facts and considerations in the previous part of the work justify. But though exceptions may be taken to not a few of the propositions stated in this book, it is without doubt a splendid contribution to the literature of cerebral disease, and will take its place as a standard work of reference among all who have to deal with diseases of the nervous system.

DAVID FERRIER.

The Crayfish. An Introduction to the Study of Zoology. By T. H. HUXLEY, F.R.S. C. Kegan Paul, 1880. International Scientific Series XXVIII.

THE publication of this carefully prepared and admirably illustrated volume should be a subject of congratulation to a large number of different kinds of readers. It will supply a need which the higher class of so-called general readers have often felt;[1] an exposition of the current theories of morphology and zoology, so illustrated by a reference to concrete facts as to enable the reader to pass his own judgment on the character of the explanations offered; in some such category as this we find, too, the medical practitioner whose knowledge of comparative anatomy has grown somewhat hazy, although he still

[1] A class which may, in Mr. Mark Pattison's words, be said to have for their aim "to improve faculty, not to acquire knowledge" (see his just published 'Life of Milton,' p. 210).

retains an affection for the science which throws so much light over the organism with which it is his especial duty to deal. The serious student, commencing morphology and acquainted with the first elements of dissection, will find that for him its chief value lies in the way in which zoological facts and anatomical characters are used to illustrate the processes by which modern naturalists attempt to explain the phenomena of living creatures. The researches of Mr. Ward, to which it is purposed to call especial attention later on, will suggest that the commencing physiologist, unknown to fame, and prevented, by the ill-directed activity of the Legislature, from experimenting on the vertebrated animals, may find in this volume not only many suggestive hints as to the physiological processes of the crayfish, but, further, that groundwork of anatomical details which will equip him for the work of resolving some still pressing physiological problems by the aid of this quite sufficiently complex organism.

There is no need to say that the book is well written, the style concise, the expositions clear; but it is a satisfaction to add that there are no attempts at fine writing or any humorous interpolations, while the woodcuts are eloquent of the care that has been bestowed upon them.

Commencing with an account of the 'Natural History of the Common Crayfish,' the author insists, at the outset, on the fact that "science is common sense at its best; that is, rigidly accurate in observation, and merciless to fallacy in logic;" this is urged from several points of view; it is shown how a common knowledge of nature grows into physical science, and how its various stages of growth can be daily exemplified from our knowledge of those around us; it is used as an argument for technical terms, and we would recommend those, who look upon science as a mere matter of hard names, to read what comes before and what succeeds the following sentence:

"Existing signs may be combined in loose and cumbrous periphrases; or new signs, having a well-understood and definite signification, may be invented. The practice of sensible people shows the advantage of the latter course; and here, as elsewhere, science has simply followed and improved

upon common sense." Whatever be the common sense possessed by those to whom this quotation is addressed, they must be careful lest they are not outstripped by the subject of this volume; we learn that not only are crayfishes intolerant of great heat, but that they affect those parts of a river which run north or south, because they yield more shade from the noonday sun than those parts which run east and west. In the winter they form burrows in the banks, and these burrows are deeper when the waters are liable to freeze. It seems, however, that from the human point of view, at any rate, the crayfish carries his common sense a little too far, perhaps because he cannot convert it into science; "crayfishes are guilty of cannibalism in its worst forms; and a French observer pathetically remarks that the males 'méconnaissent les plus saints devoirs;' and, not content with mutilating or killing their spouses, after the fashion of animals of higher moral pretensions, they descend to the lowest depths of utilitarian turpitude, and finish by eating them."

To come to the nervous system itself. As is well known, all animals which are known as invertebrata are distinguished from those which are called vertebrata (here, of course, including, as a degenerated branch, the tunicata) by the obvious fact that their central nervous system is "ventral" and not "dorsal," or, in other words, that they are "neuropodous" and not "hæmapodous"; the "ventral ganglionic chain" is in animals which, like the crayfish, are made up of a number of successive segments arranged typically in the same metameric fashion as the appendages and some other of the organs of the body; it is, however, rare to find in the adult that the masses of ganglionic matter retain their primitive disposition; some of the lowlier crustacea would appear to do so, but in all the higher forms there is apparent, especially in the more anterior region, a more or less striking concrescence of a certain number of ganglionic masses; in a common crab (*Carcinus mænas*) this fusion is carried, for example, to an extreme, and the whole of the ganglia of the ventral cord are fused into a single mass. The crayfish has thirteen distinct ganglia to its twenty segments, and of these the first is the brain or supra-œsophageal mass; the second or sub-œsophageal ganglion is

apparently composed of five primitively distinct masses, for it sends off nerves for five segments, while the supra-œsophageal ganglia are, as in so very many of the forms allied to the crayfish (other arthropoda), formed by the fusion of three others.

These ganglia are connected together by a pair of commissural cords of varying length, while they give off a number of fibres either to the muscles, or to the organs of sense, or to the integument generally; and the only important difference between them seems to lie in the influence which the supra-œsophageal ganglia have over the rest. When these are extirpated we find that, as Professor Huxley states, the creature, which, when uninjured, always attempts and ordinarily succeeds in regaining its proper position if it be placed on its back, is now no longer able to do so; "its limbs are in incessant motion, but they are 'all abroad;' and if it turns over on one side, it does not seem able to steady itself, but rolls on to its back again." If, previously to the operation, anything is put between its own chelæ it may try to use it as a means to turn over; but after extirpation of the ganglia it seizes the object and tries to swallow it; if the object is pulled back, the chelæ of the other side are brought to its aid and a co-ordination of movements is strikingly exhibited; this coordination may be destroyed by an ablation of the thoracic ganglia. These observations are sufficient to show (1) the influence of the cerebral ganglia, and (2) the independence of the succeeding portion of the nerve-cord. The results attained to by Mr. Ward go very much further, and it will not be inappropriate here to draw attention to the results which, in his preliminary notice, that observer has put out.[1] The first inference to which his researches have led him is this, there is no decussation of the longitudinal fibres in the cray-fish; this dictum will be found to be supported by the fact that division of one of the supra-œsophageal commissures is followed by an enfeebled nervous activity on the same side; not only does it happen that feeble, if any, responses are given to "considerable

[1] 'Observations on the Physiology of the Nervous System of the Crayfish.' By James Ward, M.A., Proc. Royal Soc. March 6, 1879. (P.R.S. xxviii., p. 379: Communicated by Dr. M. Foster, F.R.S.) See, also, 'Journal of Physiology,' II., pp. 214–228.

excitation" of the antennæ and eye-stalks, but a conspicuous want of symmetry is observable in the action of the abdominal appendages, and "the chelæ during progression show a bias towards the sound side."

The second result is of considerable importance. It was found that the spontaneous, or "volitional" activity of the *animal as a whole* depended upon the functional presence of the supra-œsophageal ganglia. This was shown not only by the positive fact that the animal exhibits spontaneity and purpose so long as one commissure remains intact, but by the negative results observed when both are cut. These results must be quoted in Mr. Ward's own words:—

"When both commissures connecting the supra- with the sub-œsophageal ganglion are divided, everything of the kind [spontaneity and purpose] disappears, save that occasionally the antennæ are waved about in the normal fashion, though much more feebly. The animal lies on its back, the maxillipedes, the chelæ, and the first three pairs of legs, for the most part, swinging slowly to and fro in perfect *tempo;* not, however, as the swimmerets do, both sides synchronously, but with the movements of one side alternating with those of the other. . . . . The feeding movements are a perfect mimicry of the movements made when food is actually seized. These last appear to be in all respects perfectly co-ordinated; so much so, indeed, that the chelate legs will wait their turn to pass their morsel to the mouth when scraps are placed in all of them at once. But neither they, nor the chelæ, nor the posterior maxillipedes show any selective power, even the animal's own antennæ being seized. The first evidence of taste appears when the food gets within the gape of the mandibles." When, in such condition of its nervous centres, the animal attempts to walk it is found that after a few slow and tottering steps it rolls helplessly on to its back. The results just now detailed are also sufficient to show that the supra-œsophageal ganglion presides over the inhibition of the "aimless and wasteful mechanical activity of the lower centres," and over the power to maintain equilibrium.

When the supra-œsophageal ganglion has been put aside, the next question is as to the special influence which the sub-

œsophageal ganglion may possess. This, again, may be looked at from what has been called the positive and the negative aspect. As to the former, it is, as Mr. Ward points out, obvious that "the sub-œsophageal ganglion is the source of a considerable amount of motor energy," for not only is there, as already detailed, an amount of movement still possible when these centres are still connected with the posterior portions of the cord, but it was found on experiment that the posterior maxillipedes still exhibit sufficient energy to raise the creature several times, and preening movements of the hinder limbs are well exhibited; better, indeed, than before. On the other, or negative side, we find such evidence as this: "The chelæ sprawl helplessly on either side, and the legs are for the most part doubled up under the body." The chelæ, too, are not always successful in passing food to the mouth, and when they do get it there they still do not let it go. The sub-œsophageal ganglion would seem, therefore, to be the centre for the feeding movements.

Our quotations from Mr. Ward are even already not inconsiderable; but it is impossible to put into less concise language the differences between the crayfish and the frog as enunciated in his next paragraph:—

"There is much less solidarity, a much less perfect *consensus* among the nervous centres in the crayfish than in animals higher in the scale. The brainless frog, e.g., is motionless except when stimulated, and even then does nothing to suggest that its members have a life on their own account; whereas the limbs of a crayfish, deprived of its first two ganglia, are almost incessantly preening, and when feeding movements are started the chelate legs rob and play at cross purposes with each other as well as four distinct individuals could do."

Here we must leave Mr. Ward, but we cannot do so without expressing a sincere hope that a full account of his important researches will soon be published,[1] nor without returning again to the point at which we hinted at the commencement that other physiologists will concern themselves with a creature

[1] A notice by M. Yung in the 'Comptes Rendus,' lxxxviii. p. 347, affords an independent support to several of Mr. Ward's conclusions.

in which the phenomena of inhibition seem to be particularly easily accessible.

The general reader already referred to will probably be a little astonished at the heading of page 89. The ordinary human being has, for no reason at all, either positive or negative that we have ever been able to discover, except from the strength of his position as a lord of creation, assumed to himself the especial and peculiar possession of a mind; the term, therefore, "the crayfish mind" will, no doubt, excite his antipathies, and the fact that the question of the crayfish having or not having a mind is left an open question will hardly console him. The various steps of a reasoning process, and the stage at which this becomes logical are, nevertheless, most admirably stated in a few short paragraphs by Professor Huxley, and the whole question summed up by pointing out that we could only have a "positive assurance" that the crayfish possesses consciousness by being a crayfish ourselves. Seeing no expectation of this, the phenomena associated with the activity of nerve and muscle are carefully explained, and form the basis for the important physiological lesson that so-called spontaneous movements do not really cause themselves, and that what, at any rate, the crayfish "does at any moment would be as clearly intelligible, if we only knew all the internal and external conditions of the case, as the striking of a clock is to any one who understands clockwork."[1]

Some points which especially interest nerve-physiologists are, however, altogether passed over. This, no doubt, is completely in accordance with the aim which this experienced expositor of scientific facts had definitely set before himself; but it should be pointed out that, so far as analogy, at any rate, is concerned, it is a considerable advantage to the student of the vertebrata to know that there is a visceral system of nerves in the non-vertebrated animals. The ganglia on the dorsal surface of the stomach, and the two nerves given off from the œsophageal commissures, to say nothing of the plexus

[1] A sentence of Mr. Pattison's ('Milton,' p. 174) is well worthy of being quoted in this connection: "That in selecting a Scriptural subject he was not in fact exercising any choice, but was *determined by his circumstances, is only what must be said of all choosing.*"

which is reported to be connected with the last abdominal ganglion, are so far of importance that they remove a difficulty from the student, who so frequently learns nothing of a sympathetic system till he comes into contact with vertebrated animals, and they seem from their origin to have a still higher significance, if the view to which Mr. Balfour is evidently inclined shall be justified by further investigations; the view, namely, that intestinal branches are developed on the main nerve stems of the thoracic and abdominal portions of the central nervous system, and that the ganglia developed on these become connected by a longitudinal commissure.[1]

This, of course, would be by the way in any other journal than 'BRAIN.' Having said this, we may, however, draw attention to one or two interesting points in nerve-muscle physiology which have recently resulted from the observations of continental observers:—

(1) When the visceral nerves passing off from the commissural cords are directly excited, the movements of the heart are accelerated; but if the thoracic ganglia (which communicate through the supra-œsophageal ganglion) are excited, the heart's beat is retarded (Yung). These first results may, perhaps, be not unfairly compared with the influence of the spinal *accelerator* nerves in the vertebrata.

(2) The tail-muscles of the crayfish soon lose their power of contraction when they are rapidly affected by a number of electric shocks; this is in keeping with the fact that the animal never swims for long at a time. (Richet.)

(3) The muscles of the great chelæ may retain their contractility for as long as four days after separation from the body; this, too, is to be correlated with the creature's well-known habits. (Richet.)

(4) Motor excitations pass more slowly along a lobster's motor nerve than along that of a frog; the proportion, per second, being six to twenty-seven mètres. (Frédéricq).

These, and observations like these, which might assuredly be multiplied, are, from a general point of view, of deep significance as illustrating, just as much as every series of

[1] 'Elasmobranch Fishes,' p. 173.

investigations into morphology or physiology does, the deep truth of Goethe's words:—

"Alle Gestalten sind ähnlich, doch gleichet keine den andern."

And they lead us, as they ought to lead us, to examine with respectful attention that theory of Evolution which Professor Huxley has done so much to render intelligible as well as to demonstrate as reasonable.

Let us now see how he deals with what he finds in the crayfish to support the doctrine he has for many years, through good report and ill, so manfully defended. No division of the animal kingdom is, in this connection, more interesting than the crustacea; for from no group have more convincing argumentations in favour of the hypothesis of Evolution been drawn. It is now some sixteen years since one of the most philosophical and careful of modern naturalists, Fritz Müller, tested the views of Mr. Darwin by an especial reference to the characters of the crustacea. His essay, entitled 'Für Darwin,' has been translated into English, and is continually in the hands of those who are interested in the discussion. As compared with his work, which to the student is of inestimable value, the contribution now offered by Professor Huxley is especially interesting. Fritz Müller's work was written in the days now fortunately quite gone by, when the attacks on the great modern prophet of Evolution were of a character which, justly enough, roused the spirit of his disciples. In the discussion under that aspect Professor Huxley took his share; but in the present volume we learn even more convincingly than we do from those remnants of the past style of "anti-Darwinian" criticism which now and again appear in our monthly magazines that the battle is really over, and that, till some formidable rival of greater scientific pretensions shall appear, the doctrine of Evolution has gained the day. In earlier times Mr. Huxley has known how to use all the weapons of scientific controversy; to-day he has found it safe to lay aside all armour, and, like his master in this great matter, he has dared to expose even his weak points. The discussion, therefore, to which he invites his readers in the last chapter of the volume is not only interesting from the way in which difficulties are

pointed out, but it is valuable as affording evidence that in matters more or less hypothetical the naturalist now, as in the pre-theoretical days which some still profess to admire, has no other aim than that of coming, after a time of struggle, to some clearer apprehension of the real meaning of natural phenomena. It will be right here to quote our author:—

"The preceding discussion must rather be regarded as an illustration of the sort of argumentation by which a completely satisfactory theory of the etiology of the crayfish will some day be established, than as sufficing to construct such a theory. It must be admitted that it does not account for the whole of the positive facts which have been ascertained; and that it requires supplementing, in order to furnish even a plausible explanation of various negative facts." (p. 333.)

The positive fact here referred to is this. So far as we know their characters, the East-American genus (*Cambarus*), in which there is one gill less than in our crayfish, has among the crayfishes (*Astacus*) its most closely allied form in the crayfish of the Amurland and of Japan; while the West-American forms are more like the Ponto-Caspian form. No information that we have at present of the disposition of land and sea in earlier geological epochs can yet explain this difficulty. An explanation of the negative fact that crayfishes are not found in certain regions is afforded us by the idea that physical obstacles, of which we have no knowledge, prevented the primitively marine ancestor from ascending certain rivers, and by the not unlikely suggestion that in certain rivers the struggle for existence was more than the invader could sustain.

Our space does not permit us to follow the author into his account of the fossil forms allied to the crayfish; into the accurate and instructive definition which he gives of the meaning of those difficult abstractions, species and genus; or into the history of the development of the special form under description. It will be interesting, however, to draw attention to a single example, which will, perhaps, suffice to show that while embryologists are accused of weaving phylogenies without end, they are, in truth, beset with many difficulties, the partial resolution of which has at least to be accomplished before they can attain to any consistent theory of develop-

ment. As in many other forms, part of the large intestine of the crayfish and of the lobster is formed by an inpushing of the ectodermal lining of the body; in the lobster it is estimated that one-sixth, and in the crayfish fifteen-sixteenths, of the large intestine have their origin from the outer layer of the body. Differences such as these are continually met with by the student of development; they are discussed with his co-workers, and it is the grand results of continuous and comparative labours which the world is apt to denominate as the results of a superficial examination. The more deeply we study the animal world, the more do we see how apparently interminable these differences are. They are hard enough to cause at times a feeling of despondency, which is only relieved by a generous sympathy; this naturalists have often, though hardly often enough, extended to their fellows. Now, however, we shall be able to look to a wider circle of friends; for no intelligent man or woman will read 'The Crayfish' without having excited in them a deeper interest than they have yet felt in the details of animal organisation, and in the labours of its investigators. From this important point of view the work now in hand is especially to be welcomed.

F. JEFFREY BELL.

Pseudo-hypertrophic Muscular Paralysis. A Clinical Lecture. By W. R. GOWERS, M.D., F.R.C.P. London: J. and A. Churchill, 1879.

THIS volume contains a reprint of a lecture which appeared in the *Lancet* for July, 1879, with the addition of some further cases and details. It presents a careful summary of our present knowledge of the clinical history and pathology of this disease, to which the author has made valuable contributions. Dr. Gowers believes that the change in the muscles is a morbid growth of connective tissue and fat in the place of striped muscular fibre, and he regards the condition as essentially a perversion of development. The changes in the spinal cord which he and the late Dr. Lockhart Clarke described were slight, and he believes were probably secondary, as many com-

petent investigators have in other cases found the cord normal; where more definite changes—e. g. sclerosis of lateral columns, atrophy of nerve cells—have been found, he thinks there is reason for believing that some other pathological state has been confounded with this disease. In detailing the clinical history, which is founded upon an analysis of 220 cases, 19 of which have come under his own observation, Dr. Gowers directs attention to the distinctly hereditary nature of the affection, in some instances six or even eight members of the same family having suffered from it; to its tendency to affect males rather than females, in the proportion of 6 to 1; to its incidence at an early age, usually before six years. He describes the insidious manner in which the disease reaches development; weakness of the knees, and slight unsteadiness of gait being often for some time the only facts observed, and as often little regarded. The *enlargement* of the muscles, which has been so much insisted upon, may be absent, and affects as a rule only certain groups, notably those of the calf, the infra spinati, deltoids, and triceps; *wasting* of the muscles is quite as common, but affects different groups, especially the latissimi dorsi and pectorals. But both enlarged and diminished muscles are weak, the symptoms of the disease are the result of this weakness, and Dr. Gowers lays great stress upon the efforts made by the little patients to raise themselves from the ground, by grasping their knees with their hands as a peculiar movement quite pathognomonic in its significance. The treatment recommended is chiefly to improve the general nutrition by arsenic and cod-liver oil, and the muscular nutrition by faradism and moderate gymnastic exercise, aided by friction; but unfortunately little can be hoped for, as scarcely an authenticated cure has been recorded, the patients becoming emaciated, their respiratory muscles failing, and death finally resulting from some acute or chronic pulmonary disease.

Dr. Gowers has increased the value of his work by an appendix giving notes referring to several points of interest, by a copious bibliography, and by a lithographed plate of the microscopical appearances of the diseased muscles.

ROBERT SAUNDBY, M.D.

The Index Medicus. A Monthly Classified Record of the Current Medical Literature of the World. Compiled under the supervision of DR. JOHN S. BILLINGS and DR. ROBERT FLETCHER. New York, F. Sypfyoldt. London, Trübner & Co.

IT is impossible to exaggerate the utility of this work, or to extol too highly the accurate industry of those who are engaged in its compilation. Recording as it does all new publications in medicine, surgery, and the collateral branches of science, and all original communications in medical journals, and transactions of medical societies, it arranges these in a manner that renders reference easy, the nomenclature and classification adopted being those of the Royal College of Physicians of London, based on Dr. Farre's well-known system. It brings compendiously before the worker in each department all contemporaneous research and speculation in his own field; it catalogues all substantial additions to medical knowledge, and it provides an inventory of the passing medical fashions of the day. To all medical practitioners, teachers, and authors it must prove useful, but to the provincial student, who has not access to medical libraries and their array of journals, it will be especially valuable, by enabling him to ascertain what is being done by others in any subject that he may be investigating, thus guiding and stimulating his explorations, and saving him from the repetition of twice-told tales. Happily provincial students of medicine—that is to say, those medical men who are not content with the routine of practice and money-making, but who feel the obligation under which they lie to promote the progress of medicine—are daily increasing in number. The country districts furnish much admirable work, and we think that the 'Index Medicus' will tend to improve the quality of this work and increase its quantity, while at the same time it diminishes its bulk.

J. CRICHTON-BROWNE, M.D.

Skin Diseases, including their Definition, Symptoms, Diagnosis, Prognosis, Morbid Anatomy and Treatment. A Manual for Students and Practitioners. By MALCOLM MORRIS. London. Smith, Elder, & Co., 1879. 8vo, pp. 288.

THE author of this manual has evidently a full and intimate acquaintance with the literature of dermatology, and with the most recent developments and appliances of cutaneous medicine. He has produced a plain practical book, by aid of which, who so chooses, may train his eye to the recognition of slight but significant differences. The descriptions are neither too vague nor over-refined; the directions for treatment are clear and succinct. We cannot add, however, that Mr. Morris has surmounted the everlasting stumbling-block of classification, his system being compounded out of those of Erasmus Wilson and Hebra, with original touches here and there. Under the class Neuroses we find zoster, cheiro-pempholyx, pruritus, and dystrophia cutis, while urticaria takes place under the Erythematous Exudationes, pemphigus under the Vesicular Exudationes, morphœa under the Hypertrophiæ and alopecia areata under Diseases of the Hair. An appendix of formulæ for baths, ointments, lotions, &c., would much enhance the value of the book.

J. CRICHTON-BROWNE, M.D.

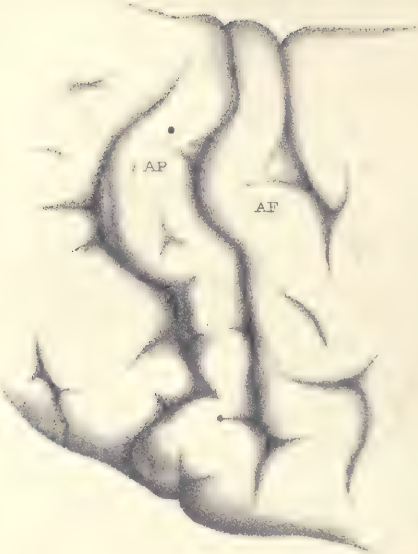
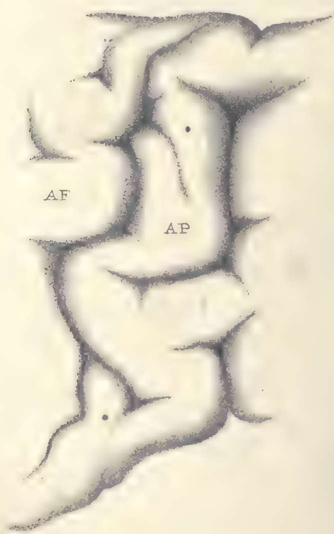


Fig. 1. (Right)

Fig. 3.

Fig. 2. (Left.)



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Clinical Cases.

ARREST OF DEVELOPMENT IN THE LEFT UPPER LIMB, IN ASSOCIATION WITH AN EXTREMELY SMALL RIGHT ASCENDING PARIETAL CONVOLUTION.

BY H. CHARLTON BASTIAN, M.D., F.R.S.,

and

V. HORSLEY,

Assistant-Demonstrator of Anatomy in University College, London.

In the number of 'Brain' for October 1878, Dr. Gowers has published an account of a case in which congenital absence of the left hand co-existed with unequal development of the ascending parietal convolutions, the right being the smaller. Towards the close of 1879, a case, which has since proved to be very similar, was admitted into University College Hospital, under the care of Dr. Bastian. The following facts concerning this case seem to us worthy of being recorded.

J. M. B., aged 26, a clerk, was admitted into the hospital on October 18th, 1879, suffering chiefly from mitral obstruction and regurgitation. He got rapidly worse (owing in part to rupture of one of the chordæ tendineæ of the mitral valve), and died rather suddenly on October 30th, 1879.

Necropsy, Oct. 31st, 1879. The body, on the whole, showed slight muscular development, especially in and in relation with the left upper limb, both pectoral muscles on that side being absent. Unfortunately, permission to dissect the limb (which was shorter and much slighter than that of the right side) could not be obtained, but the arrangement of the bones in the small and abortive "hand" was readily felt through the skin, and is represented in the accompanying figure (H), copied from a sketch made by the physician's assistant, Mr. B. Pollard. The dotted lines show the outlines of the bones. The position and proportion of the metacarpals bear a close resemblance to the early appearances of segmentation, seen as slight ridges and intervening depressions in the forelimb of an embryo (man)

about 2 cm. in length. In the case of Dr. Gowers, above referred to, in which one of us had the opportunity of dissecting the limb, the theory of intra-uterine amputation was favoured by the condition of the parts; but in the present instance the appearances are strongly in favour of arrest of development.

The brain, on removal, was divided longitudinally, and each half placed on its inner surface in a 2·5 per cent. solution of bichromate of ammonia. The membranes were at once removed over the ascending convolutions on each side, when a striking difference appeared (as shown in figs. 1, 2). After 24 hours, the middle three-fifths of each ascending parietal convolution was measured at seven equidistant points between the dots in each figure, and the following results were obtained:

	Left A.P.C.	Right A.P.C.
	cm.	cm.
1	1·4	1·5
2	1·6	1·5
3	1·5	1·85
4	1·4	1·7
5	1·3	·8
6	1·9	1·0
7	1·8	1·3

Thus it is seen that at the points 5 and 6, the difference in breadth was almost 2 to 1 in favour of the left asc. par. conv., and that, on the whole, there was a difference of ·2 cm. between the right and left convolutions.[1] There was no such striking asymmetry in the rest of the brain; the basal ganglia and descending motor tracts in the pons and medulla being apparently equal. A careful microscopical examination revealed no appreciable difference in the internal or minute structure of the two convolutions.

From the above facts we see that the regions of the cortex, corresponding with the "centres" "*a, b, c, d*" of Dr. Ferrier,[2] were but slightly developed on the right side. The actions evoked by stimulation of these centres he gives as, "Individual and combined movements of the fingers and wrist, ending in clenching of the fist." And in this man such movements had certainly been for the most part impossible with the left "hand."

It would seem, then, that we have here another instance tending to corroborate the view that there is a correlation of some kind between the functional activities of these regions of

[1] The drawings were obtained by taking impressions of the sulci by means of tinfoil. The deepest portions of the sulci being marked by punctures, the tinfoil was laid on the paper, and the latter marked through the holes. The accuracy of the drawings was finally verified by numerous comparative measurements. The actual breadth of the convolutions themselves was filled in from the above measurements, and the figures were then reduced one-third.

[2] 'Functions of the Brain,' pp. 148 and 307.

the ascending parietal convolution, and the movements of the opposite hand and fingers—as indicated by the experimental observations of Dr. Ferrier.

It seems something more than can be accounted for by mere chance coincidence, when precisely the regions of the cortex indicated by him are found to be defective in bulk in two consecutive cases of absence or arrest of development of the hand. Yet those who are still a little sceptical will doubtless await further coincidences of the same kind, and will seek to learn something definite as to the degree of frequency with which a marked asymmetry of the ascending parietal convolutions of the two sides is to be met with in cases where no malformation of one hand is present.

The question of the existence or not of a correlation of this kind is one which does not by any means necessarily associate itself with the view that the particular regions of the cortex indicated contain "motor centres." A correlation may really exist between the activities of special cortical regions and the performance of particular movements, and yet the commonly received explanation of such a correlation may not be correct.

Thus it is possible to accept as a fact the existence of such correlations, and at the same time wholly to reject the view that anything like a "motor centre" is to be found in the cerebral cortex.

A complex group of ingoing impressions follows or is produced by movements of the hands, as of all other parts of the body, so that we may be said to have a "sense of movement" growing out of the repetition of what one of us has termed kinæsthetic impressions[1]—just as our sense of sight is developed from the repetition of visual impressions.

Sensory impressions of both these types, derived from movements, will subsequently, when ideally revived, co-operate as factors in the reproduction of similar movements. The activity of the "kinæsthetic centres," more especially, would thus be closely related to the reinitiation of movements—and different parts of such centres would have to do with the production of different kinds of movements. Thus stimulation of certain sensory regions of this type may lead to the manifestation of certain movements, whilst destruction of the same sensory regions may abolish the possibility of performing such movements. The so-called "motor-centres" may, therefore, in reality, be sensory regions of the kinæsthetic type, and these cortical areas may in addition take part in the performance of other cerebral functions whose nature cannot be at present specified.

[1] 'The Brain as an Organ of Mind' (1880), pp. 543 and 522.

The fact that the small right ascending parietal did not differ in its microscopical characters from the well-developed left ascending parietal convolution, is not incompatible with the existence of a correlation of some kind between these parts and certain movements of the hand, whether their activities be of the sensory or of the motor type. In such a case as we have now brought forward, there would be no reason to look for atrophied nerve cells, merely because certain movements have never occurred, but rather for a numerical inferiority of nerve cells and a mere inferior bulk of convolution in relation with an abortive limb whose possible movements were notably curtailed.

CASE OF NERVE-SPLITTING.

BY SURGEON-MAJOR K. M'LEOD, A.M., M.D., F.C.U.,

Professor of Surgery, Calcutta Medical College.

C. R., æt. 26, a spare East Indian youth of nervous temperament, consulted me on the 27th of December regarding a wasting of the left forearm and hand, and numbness of the latter, from which he had suffered for 8 years, and for which he had been subjected to various plans of treatment, without gaining relief from any. In the year 1871 he began to experience peculiar sensations in the little finger and ulnar side of the left hand. The parts gradually got numb, the muscles of the hand wasted, and the fingers became permanently bent. He sought relief at the Presidency General Hospital, when it was discovered that the ulnar nerve had undergone thickening. Blisters were applied along the course of the nerve, and subsequently iodine and magnetic electricity, but no benefit was derived from these energetic remedies. An abscess formed in 1875 above the elbow on the inner side, which discharged spontaneously, and left a sinus which continued to emit matter for some time, and then closed of itself. This sinus reopened in 1879, without apparently any fresh accession of inflammation. His condition when I examined him was as follows:—

Left arm generally less muscular than the right; circumference of left forearm 1 inch less than right. Special wasting over position of flexor carpi ulnaris. Hand very much wasted, more particularly the short muscles of the little finger, the interossei, and the adductor pollicis. The short muscles of the thumb are smaller in bulk than on the opposite side. The fingers are habitually bent, more especially the ring and little fingers, which are permanently flexed at both phalangeal joints. The thumb can be straightened completely, the fore and middle fingers almost completely, the ring and little fingers very partially, the first phalangeal joint remaining bent at a right angle. The action of the common extensors and flexors is unimpaired. The power of adduction and abduction of the fingers is almost

abolished. The thumb can be adducted and abducted. Sensation is abolished all over the little finger, and over its metacarpal bone on the dorsal and palmar aspect as far as the styloid process of the ulna. Sensation on the ulnar side of the ring-finger is diminished, and on the radial side perfect. Sensation over the ulnar two-thirds of the dorsum of the hand (as far as the line of the metacarpal bone of the middle finger) is very much impaired. On the palmar side sensibility extends as far as the metacarpal bone of the ring-finger. The degree of sensibility was tested by pricking with a pin, and the patient, who is a very intelligent young man, gave very prompt and definite indications of the sensations experienced.

The skin of the insensible area is somewhat congested, but there is no vesication or breach of surface. There is a hard knot on the dorsal branch of the radial nerve about 3 inches above the wrist. There is a similar knot on the great auricular nerve of the left side as it crosses the sterno-cleido-mastoid muscle, and on the frontal nerve of the right side. All these knots are painful on pressure. The ulnar nerve is very much thickened behind and above the elbow to the extent of about 5 inches. It is hard and cartilaginous to the feel, as thick as the middle finger, and painful on manipulation. There is an orifice of a sinus over the course of the nerve situated about 4 inches above the inner condyle of the humerus.

I advised the patient to submit to the operation of stretching the ulnar nerve as the only expedient likely to benefit him, and gained his ready consent.

Operation.—After bringing him under the full influence of chloroform, and washing the parts thoroughly with carbolic lotion, I passed a director into the sinus, and found that it passed into the interior of the nerve as far as the level of the inner condyle. I slit the sinus up throughout its whole extent. A long linear incision, exposing the nerve for a space of 4 inches, was the result. It was hollowed out by an abscess of which its thickened texture constituted the wall. This cavity was filled with curdy material, and stuff of the same kind escaped on pressure from orifices in the abscess wall. These orifices led to small chambers or recesses in the mass of the thickened nerve. The cavity was thoroughly emptied, and the contents of the flask-shaped recesses squeezed out. The lining membrane of the main cavity was then scraped off, and the smaller cavities carefully cleaned out with a small scoop. The nerve was then split in two from before backwards, and the division was prolonged into the comparatively sound nerve above and below, the knife following as far as possible the direction of the fibres. The continuation of the nerve behind the condyle was somewhat thickened, and contained a few cells

full of yellow curdy material. The operation was performed under strict antiseptic precautions, and the whole line of wound was very carefully brought together by iron wire and horse-hair stitches, after a few threads of carbolised catgut had been laid in it for drainage. Boracic-acid ointment, spread on muslin, was applied next the wound, and the ordinary carbolic gauze-dressing placed so as to reach to the axilla above and half-way down the forearm below. The dressing was removed every second or third day, according to circumstances. Convalescence was satisfactory. An attack of ague occurred during the second day, which yielded to quinine. The neighbourhood of the wound was intensely sensitive for the first ten days or so. The wound remained sweet and healed kindly. The following notes were taken on the 21st of January, 1880—24 days after the performance of the operation :—

"Cicatrix completely healed. No abnormal sensibility in the neighbourhood of the wound. The thickened nerve is still perceptible beneath the cicatrix, but it is not so hard or bulky as before the operation. There is no evidence of deep matter nor sinus. Above the cicatrix the nerve is slightly thickened and very sensitive; below the cicatrix the nerve is also somewhat thickened and sensitive, but less so than above. The flexor carpi ulnaris is still atrophied. He straightens his little and ring-fingers rather better, and separates his fingers with more force and effect. The muscles of the hand are still very much wasted, and the tendons very prominent. There is still slight congestion of the dorsal surface of the ungual phalanx of the little finger. Painful sensation is elicited by the prick of a pin all over the hand, except over a narrow strip on the dorsum of the little finger, as far as within half an inch of the styloid process. Pinching with the finger and thumb, however, produces pain up to the first phalangeal joint; beyond that there is no sensation, or perception of pricking, or pinching, but when the end of the finger is forcibly squeezed, pain is caused."

Remarks.—The condition for which the operation above described was resorted to is, as far as my experience goes, a very rare one. I have frequently seen thickened nerves leading to anæsthesia and impaired motility over the area supplied by them; but I have never before seen a nerve hollowed out in this fashion by an abscess, and the seat of curdy deposits. In this patient three nerves were the subject of thickening, and two of them of abscess and sinus. The ulnar nerve appears to be specially liable to this thickening. Dr. Laurie, acting on a suggestion which I threw out in 1877, stretched this nerve in 30 cases in which thickening and anæsthesia existed ('Indian

Medical Gazette,' vol. xiii., pages 229, 270), and in all of which the operation produced benefit. In two cases, regarding which he has been able to obtain information, marked and permanent improvement was experienced; and in a similar case, which I reported at length in the August (1879) number of the same journal, restoration of sensibility and power followed very speedily upon the stretching of the thickened nerve. In the present instance stretching was not resorted to—simply laying open and splitting the nerve—and although the patient did not recover sensibility or muscular power fully, the notes of the condition before and after the operation leave no doubt that very considerable amelioration was caused by the operation in both respects. How the operation of mechanical stretching restores the function of the nerve I am not prepared to say. The case now recorded would render it probable that amendment is produced by removing tension and pressure upon the nerve fibrils. The case is also interesting in demonstrating the function of the interossei and lumbricales muscles in extending the two terminal phalanges. Their tendons join the extensor aponeurosis on the dorsal surface of these phalanges. There was no impairment of power of the common or special extensors, but the paralysis of the interossei and lumbricales caused permanent flexion of all the fingers, and when the function of the ulnar nerve was partially restored, the fingers could be better straightened.

CASES OF INTRACRANIAL TUMOUR.

BY JAMES ROSS, M.D.,

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Case II.—Samuel Holmes, æt. 7 years, presented himself as an out-patient at the Southern Hospital, Manchester, on January 26th, 1876.

The following history was elicited from the mother:—He was a bright, intelligent, and healthy boy until about 15 months ago, when he fell from a wall, 5 feet high, and raised a lump on his forehead. Soon afterwards he complained of constant headache, chiefly confined to the forehead. The top of the head was also so sensitive that combing his hair caused him much pain. He could not keep still; his legs, especially, were constantly moving, and at meal times he was in the habit of knocking the table with his right hand, as if from impatience. About nine months ago the mother noticed that his mouth was slightly "crooked," and that his left arm hung helplessly by his side. The forearm was twisted so that the palm of the hand was directed outwards and the thumb backwards, his fingers were bent, but she thinks his thumb at first was held straight and drawn away from the fingers. After some weeks, however, the thumb became bent inwards under the index-finger, and she had to pare the nail of the thumb frequently to prevent its cutting the skin of the outside of the middle finger. He now began to drag the left foot in walking, and the forearm was gradually drawn up behind his back, instead of hanging, as at first, by his side.

The mother had nine of a family, no miscarriages and no still-born children. One child died from convulsions during teething; a second child, who was weakly from birth, died at three months of age; and a daughter has suffered for the last two years from white swelling of the knee.

On presenting himself at the hospital he was a well-made and fully-developed boy for his years. His head was large, but well-proportioned; face round and plump, although pallid; his incisor teeth were regular, his nose was well formed, the

muscular system was well developed, and there was abundance of subcutaneous fat. There was very well-marked left facial paralysis, so that the left corner of the mouth could not be moved in the slightest. Both eyes could be closed; the pupils were large, equal, sensitive to light, and there was no affection of the special senses. The left elbow was kept a little behind the mesial plane, and 2 inches from the side; the forearm was bent at right angles to the arm, and drawn behind the body; the hand was strongly pronated; the thumb was adducted, and the second phalanx flexed, so that the point rested against the second phalanx of the middle finger. The first phalanges of the fingers were extended and in a line with the metacarpal bones, and the second and third phalanges were flexed. A considerable amount of muscular rigidity was induced on attempting passive motion at the elbow and wrist joints. By a voluntary effort he could raise his elbow to nearly the level of the shoulder, and bring the upper arm slowly forwards; but he could neither extend the forearm, produce supination, nor extend the fingers. The left leg dragged during walking, but there was no muscular rigidity, and all the movements of the leg could be separately performed. The electro-cutaneous sensibility of the left half of the body was increased, especially over the back of the left hand, and the left half of the face and side of the head. The slightest touch of the skin over the vertex of the head to the left of the middle line caused the patient to wince, and the cutaneous sensibility to pain was increased over the left half of the body generally. The other organs were healthy, and there was no albumen or sugar in the urine. He was ordered four grains of iodide of potassium three times a day; but, as no improvement took place, he was admitted into the hospital on February 28th.

March 10*th*, 1877.—He was ordered, on admission, fifteen minims of the syrup of the iodide of iron, to be taken three times a day, and the daily application of a weak constant current to the paralysed muscles and nerves. After two applications of the constant current he could extend his fingers to a slight extent, and in a few days he was able to raise his hand to the back of his head. It was observed, however, that the most marked improvement took place at the shoulder-joint; and that improvement in the movements of the forearm and hand was only to a slight extent. This improvement was of short duration, and he now looks decidedly worse than on admission. The pallor of the face is much increased; his appetite has failed; the pulse is 110, weak and irregular; and the nurse says that he has become very stupid. Ordered to be kept in bed, milk diet and a saline mixture.

March 18*th*.—Since last report he has got steadily worse, has

vomited frequently, and to-day he has been seized with general convulsions. The convulsions frequently occurred for the next two days, and he did not recover consciousness in the intervals, and died early on March 21st.

Sectio cadaveris, twelve hours after death.—On opening the skull, the convolutions of the brain presented a flattened and compressed appearance, and about 2 ounces of fluid escaped during removal. The brain weighed 51 ounces. On slicing the brain to a level with the corpus callosum the upper surface of a tumour was exposed, which was situated in the centrum ovale of the right hemisphere, immediately to the right of the corpus callosum and at the junction of the anterior and middle lobes. On opening the lateral ventricles, this tumour was felt as a hard nodule, slightly projecting into the right lateral ventricle, and occupying the position of the caudate nucleus and anterior portion of the optic thalamus, and only covered by the ependyma of the ventricle. The tumour measured three-quarters of an inch in the transverse and an inch in the antero-posterior and vertical diameters respectively, so that not only the caudate nucleus and anterior portion of the optic thalamus, but also the anterior two-thirds of the internal capsule and the anterior portion of the lenticular nucleus were destroyed by it.

The growth was pretty sharply defined from the surrounding brain-tissue, and on section it presented an outer grey, somewhat vascular cortex, about two lines thick, and a central core of a yellow colour, and apparently destitute of any structure.

Microscopic examination showed that the grey cortex of the tumour consisted of giant cells, each surrounded by lymphoid cells imbedded in a fibrillated reticulum.

The right lung was closely adherent to the chest wall and to the diaphragm. The lung itself was congested, but every portion of it floated in water. No tubercles nor cheesy glands were discovered, and the other organs were healthy.

Remarks.—The progressive character of the symptoms in this case, as well as the fact that its origin dated from a fall on the head, pointed from the first to the growth of an intracranial tumour. My diagnosis, therefore, was tumour, probably tubercular, of the right corpus striatum. At that time I was not aware of the full significance of the fibres which pass through the internal capsule to reach the cortex of the brain. Looking back on the case now, I think it probable that the tumour began first to grow from one of the arteries which branch from the middle cerebral artery and pass through the anterior perforated space to reach the caudate nucleus of the corpus striatum. It has occurred to me that the thumping

movement of the right hand described by the mother might really have taken place in the left hand, and that these, along with the other restless movements, might have been due to primary irritation of the left caudate nucleus or the *nodus cursiosus* of Nothnagel. This supposition, however, is too doubtful for any reliance to be placed upon it. The paralysis is to be explained on the supposition that the fibres of the middle third of the internal capsule, and which connect the cortex of the brain with the spinal cord, were gradually compressed by the progressively increasing size of the tumour. These fibres were therefore compressed from before backwards, and it is important to notice that the left half of the face was more paralysed than the upper extremity, and the upper much more paralysed than the lower extremity.

The peculiar position occupied by the forearm and hand is also interesting in connection with the gradual compression of the motor tract in its passage through the internal capsule. The sensory disturbances were probably due to irritation of the fibres of the posterior third of the internal capsule. Veyssière has proved that section of these fibres in dogs is followed by hemianæsthesia of the opposite side, and the cases recorded by Dr. Hughlings-Jackson and others show that a lesion of the posterior portion of the optic thalamus, involving the posterior fibres of the internal capsule and the external geniculate body, is attended by hemi-anæsthesia of the opposite side along with hemiopia. It may, therefore, be inferred that irritation of these fibres will cause hemi-hyperæsthesia of the opposite side.

CASE OF ALTERNATE HEMIPLEGIA, WITH CONJUGATE DEVIATION OF THE EYES.

BY ALEXANDER M. M^C^ALDOWIE, M.D.,

Resident Medical Officer, North Staffordshire Infirmary.

IN March 1876 I was sent for to see John S., aged 29, a labourer, stated to be suffering from a "stroke of paralysis."

The following history was elicited:—

The patient contracted syphilis eight years ago, and suffered from ulcerated throat and skin eruptions for several years subsequent to the chancre, but has been free from these complaints for more than two years. Has been troubled with pains in the shins and arms for nearly six months, and from dull aching pains in the occipital region, together with giddiness, almost every night for the past three months. Four days ago the patient suddenly lost the use of the right arm and leg while dressing himself in the morning. There was no loss of consciousness at the time of the attack, although the patient felt giddy and his eyesight grew dim. The power of speech was lost for some hours. Patient had felt no premonitory symptoms.

Present condition.—Patient well nourished. Intellect very good. Complains of severe pain in the occipital region. Pain heavy and dull in character, most intense during the night. Total paralysis of the upper, and almost entire loss of power over the lower, extremity on the right side. (Patient states that the paralysis of the leg was complete for two days after the attack.) Sensation greatly diminished in the paralysed limbs; electrical excitability impaired; sensation of heat and cold normal. Complete paralysis of the left side of the face. Tongue protruded straight. Speech thick and very indistinct, but no aphasia. Ptosis of left eyelid; slight twitching of right upper eyelid. Marked deviation of both eyes to the right, the patient being unable to turn them towards the left beyond the middle line. Pupils natural in size, react readily to stimulus

of light. Ophthalmoscopic appearances normal, both discs pale, outline well defined, vessels natural. Nothing abnormal detected on examination of the chest and abdomen.

Ordered 10 grs. of iodide of potassium three times a day.

The patient improved steadily under treatment. The deviation of the eyes disappeared about three weeks after the attack, but the facial paralysis lasted for nearly two months. He regained the power of the leg so as to be able to walk fairly well. The arm remained very weak, the patient being able to use the hand and forearm, but was unable to raise the limb to the horizontal position.

The treatment was anti-syphilitic throughout, iodide of potassium being administered at first, afterwards liquor hydrargyri perchloridi, and finally biniodide of mercury.

The patient was lost sight of about four months after the attack.

Remarks.—A case of hemiplegia alternans with conjugate deviation of the eyes, is of considerable interest and worthy of record.

Alternate hemiplegia is the most characteristic symptom of disease of the lower part of one lateral region of the pons. In this case the lesion was evidently situated at the lower part of the left side, involving the fibres of the facial nerve below their decussation in the pons, and the motor fibres for the limbs above their decussation in the medulla.

The completeness of the facial paralysis observed in this case is also a prominent sign of disease in this region. Bastian says that in lesions in this part "we have an unusually well-marked facial paralysis on the side of the brain lesion, and a more or less complete motor and sensory paralysis of the limbs of the opposite side." And Nothnagel observes: "In this variety of facial paralysis of cerebral origin, all the branches of the facial nerve, even those supplying the frontalis, corrugator supercilii, and orbicularis palpebrarum, are involved, just as in the peripheral form of this affection; whereas, when the lesion is situated in the upper part of the pons, at least in the cases which I have observed, these branches escape."

The conjugate deviation of the eyes from the side of the brain lesion and towards the paralysed limbs noted in the above case, also points to a lesion of the pons. Prévost was the first to point out that the deviation of the eyes follows a constant law, being always towards the side of the brain lesion. The only exceptions that he admits are affections of the pons and medulla, when the deviation may be away from the side of the brain affected. Bernhardt argues that this symptom has

no diagnostic value, but the small number of exceptions that he brings forward only show that the law is not so constant as Prévost maintains. Taken in connection with the alternate hemiplegia observed in this case, the symptom is of importance in aiding us to localise the disease.

As to the nature of the affection, judging from the history and the result of the treatment adopted, there can be no doubt but that the disease was of syphilitic origin, probably the first form of "syphilitic new-growth" described by Heubner.

CRURAL MONOPLEGIA—LIMITED CORTICAL LESION OF OPPOSITE HEMISPHERE.

BY DAVID FERRIER, M.D., F.R.S.

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On November 18th, 1879, I saw S. W., ætat. 40, of whose case the following are chief particulars, as furnished me by Dr. Brookhouse, of New Cross, with whom I saw the patient in consultation.

The patient was first seen by him on Sept. 21st, 1879, and had been feeling "out of sorts" for a week previously with symptoms of what he called a bad cold. He had a temperature of 101° F., pulse 100, anxious expression, and some abdominal tenderness, but no diarrhœa. He had a slight cough, but there were no abnormal physical signs on auscultation and percussion.

His symptoms fluctuated between this time and October 9th, but he became weaker; his appetite became impaired, his tongue coated and flabby, and his breathing became laboured on exertion. Nothing could be detected on careful examination of the lungs except a few moist râles at the right base posteriorly. The symptoms in many respects resembled typhoid.

On Oct. 24th he complained of some stiffness in the left leg, and inability to move it so freely as the right. On the 25th it became still more feeble, and on the 26th it became completely paralysed as to motion.

This continued till Oct. 28th, when he said that he feared the left arm was "going like the leg." The grasp of the left hand was distinctly feebler than the right. Next day the feebleness was still more pronounced, and by the 31st the left arm had become completely paralysed like the leg.

During the night of Nov. 6th, he had a series of convulsive movements of the left leg and arm, commencing in the leg and lasting half an hour.

From this date the patient grew worse. His temperature

ranged between 99° and 102°·4 F., there being no marked difference between morning and evening temperatures. There was no dulness in the lungs, but fine crepitation on both sides posteriorly. There was much cough, with frothy yellowish spatum, but quite free from any rusty appearance.

On Nov. 18th I saw the patient with Dr. Brookhouse with a special view to determine the nature of the paralytic affection. The patient was somewhat flushed, breathing rapidly and with an appearance of oppression when he spoke, coughing a good deal and expectorating frothy mucus. His temperature taken on the right axilla was 102° F., while on the left axilla it was 102°·8 F.

There were no symptoms of extensive cerebral disease. The mind was clear. There was no headache or vomiting, and examination of the discs revealed nothing abnormal.

There was nothing abnormal discoverable on examination of the abdomen. The urine was free from albumen. The lungs were resonant throughout, but there were moist sounds at the base posteriorly, particularly on right side. On examination of the limbs the left leg and left arm were seen to be perfectly flaccid and powerless. There was a total loss of volitional power. There was no trace of facial paralysis. Both sides acted equally well, and the tongue was protruded straight.

There was increased tendon reflex in the left leg. Sensation was unimpaired. The slightest touch on any part of the left arm or leg was distinctly perceived and accurately localised.

The patient had had syphilis and had lately been under specific treatment, owing to suspicious syphilitic ulceration of the tongue. A sister had died of acute tuberculosis. From a consideration of the past history and symptoms presented by the patient, I formed the opinion that the constitutional symptoms and febrile excitement depended mainly on acute infiltration of the lungs, either tubercular or syphilitic, and that the paralysis depended on a limited lesion of the cortex, progressive in character, and situated at the upper extremity of the fissure of Rolando on the right hemisphere.

An unfavourable prognosis was pronounced.

No specially fresh symptoms occurred, but the patient gradually sank and died on Nov. 24th, in a state of coma.

Post-mortem examination.—On November 26th we made a post-mortem examination, but had to confine our examination to the head and partial exploration of the thorax.

The skull-cap was easily detached and was free from adhesions. The dura mater was also easily separable, except for slight, easily broken down, adhesion at the middle line at

a region corresponding to the upper extremity of the fissure of Rolando on the right side. The subarachnoid space contained some fluid, but clear and free from opacity. The pia mater was everywhere normal and separated readily from the cortex except at one spot. This was situated at the upper margin and internal aspect of the right hemisphere on both sides of the fissure of Rolando, where the pia mater merged into a caseous adhesion not appreciably elevated above the rest of the cortex, but which could not be removed without tearing the cortical substance, and causing erosion.

On further careful examination of the position and extent of the lesion, the following notes were made:—

The pia mater was adherent and studded with minute tubercular foci, of a yellowish aspect, on the internal aspect of the hemisphere at the point where the fissure of Rolando terminates. The adhesion was of a quadrilateral shape, measuring 1 in. antero-posteriorly and $\frac{3}{4}$ in. vertically. (The position and extent of the lesion here will easily be understood by reference to Ecker's figure of the internal aspect of the hemisphere. It occupied an area which would be included between the letters A and B, and concealing C.)

Doubling over the margin of the hemisphere the adhesion separated the extreme upper lips of the fissure of Rolando from each other, and descended along the bottom of this fissure, coming to a point an inch below the margin of the hemisphere. The whole area and shape of the lesion may be likened to a triangle, the base of which would measure 1 inch, and the height $1\frac{3}{4}$ inch or 2 inches at the utmost, applied with its base at the internal termination of the fissure of Rolando, and doubled over the margin with its apex directed along the bottom of this sulcus.

Removal of the adherent membrane caused tearing and erosion of the cortex, but quite superficially; the medullary substance underneath being injected but not softened.

The degeneration was most advanced internally and at the extreme upper margin of the hemisphere, as indicated by the caseous softening of the miliary tubercles.

There was no excess of fluid in the ventricles, and the rest of the brain was normal throughout.

The lungs, portions of which were removed and examined microscopically were studded throughout with miliary tubercles, feeling to the touch as if infiltrated with small shot. There were no cavities or areas of softening.

The other viscera were not examined.

Remarks.—This is an important addition to the comparatively small number of clinical cases serving to indicate the

position of the leg-centre in the cortex. It is an altogether uncomplicated instance of purely cortical lesion, so definite as almost to compare with a physiological experiment. Though the paralysis did not remain confined to the leg throughout, yet the fact that it did so for four days clearly proves that the leg-centre is capable of differentiation from those of the arm, though they are so commonly implicated together. The point where the lesion began is indicated by the region where the caseous degeneration was most advanced. This was the inner aspect and margin of the hemisphere on each side of the upper extremity of the fissure of Rolando. This situation accurately corresponds to the areas—1 and 2—which I have indicated on the monkey's brain as comprising the centres for the various movements of the leg. The extension of the paralysis to the arm is in accordance with the advance of the lesion along the fissure of Rolando.

The diagnosis—which I made with confidence from the mode of onset and its progress, the character of the paralysis, and absence of general cerebral symptoms—of a limited cortical lesion of a progressive character, situated primarily at the upper extremity of the fissure of Rolando, was completely verified by post-mortem examination. The case shows *inter alia* that with lesions strictly cortical there may be the most absolute motor paralysis, with perfectly intact cutaneous sensibility; and, further, that with cortical lesions vaso-motor paralysis is associated with motor paralysis, as indicated by the higher temperature—·8° F.—of the paralysed side.

Abstracts of British and Foreign Journals.

Innervation des Vaisseaux cutanés, Dastre et Morat. (*Archives de Physiologie*, Mai—Août, 1879.)—The above paper enters into an exhaustive critique of vaso-dilatation, and furnishes certain precise data towards the solution of its problem. The central origin of vaso-constrictor nerves is first discussed—it is now-a-days proven that they are associated with the anterior spinal roots, the dorsal division of the cord being their principal focus of origin; but there is a further centralisation of influence within a limited portion of the bulb, the general tonic centre or vaso-motor centre of Owsjannikow; and vaso-motor centres have further been assigned even in the cortex. Of the degree of centralisation and of the relative independence of an axial series of spinal centres, D. and M. remark that the universal influence of the bulbar centre is undeniable, but that this influence is not exclusive—the conclusion is that vaso-constrictor activity has one chief source (bulbar) and several accessory sources (spinal and encephalic). Still another element comes into account—the possible or probable existence of peripheral tonic centres (Huizinga) constituted by the ganglionic cells in intrinsic plexuses of the arterial wall—intrinsic arterial ganglia. One among many good arguments in their favour is as follows: a dilatator (e. g. corda tympani) contraction of the vessels by irritation of the sympathetic is hindered by simultaneous irritation of the corda; since the nerves run independently to the periphery, it is here that we must presume some mechanism to exist compounding their opposite actions. The question of dilatation is considered at length—whether it is by an activity opposed to constriction, or merely by an absence of constriction.

The definition of a dilatator nerve is insisted upon as a centrifugal nerve whose excitation effects a primary dilatation of vessels independent of any central mediation, thus excluding the confusing consideration of reflex dilatations. We are agreed as to the mecha-

nism of constriction, not so as to that of dilatation. Dismissing the notions of a direct dilatator influence of the nerve fibre on the muscle fibre, or of the dilatation of arterioles by constriction of venules, or of exaggerated peristaltis, it cannot be denied that every dilatation is a paresis of constriction, and before the suggestion of dilatators this was the universal statement; but the discovery of the dilatator action of the corda tympani led to the generalisation of active dilatation opposed to contraction, and subserved by vaso-dilatators distributed with vaso-constrictors throughout the body. Dilatators were then sought for, and under favourable circumstances for the most part found. The sciatic has been throughout a chosen bone of contention; the notorious discrepancies of results are attributed by D. and M. to inadequacy of tests. The most employed has been thermometry; less frequently, observation of a bleeding-point and plethysmography. The thermometer is unsatisfactory, because it is a sluggish test, and because it is not proved that temperature varies coincidently with dilatation; note also that in the scale of low temperatures, with constriction of vessels, dilatation is most readily manifested and its thermic effect greatest, and *vice versâ* in the scale of high temperatures; further that when vessels dilate warmth increases, and dilatation is therefore cumulative. Classing observers into: (1) those to whom the sciatic is a dilatator (Golz, Masius, Vanlair); (2) those to whom it is constrictor *and* dilatator, according to circumstances (Onimus, Lépine, Kendall and Luchsinger, Bernstein and Marchand, Grützner and Heidenhain); (3) those to whom it is a constrictor (Putzeys, Tarchanoff, Vulpian)—D. and M. remark that classes (1) and (2) employ the thermic test, class (3) have recourse to direct observation.

D. and M. resorted to manometry.—They first estimated the local variations of pressure effected by an accepted constrictor (sympathetic), with these they then compared the local variations effected by a mixed nerve (plantar branch of sciatic). The results were in either case identical. With the sympathetic, facial vein and facial artery of horses or asses, they obtained with the section of symp. (1) a brief elevation of both pressures (lasting 6″ to 8″), due to constriction of vessels by the momentary irritation of section, followed by (2) a gradual rise of venous and fall of arterial pressure. With tetanisation of the symp. they obtained (1) a brief rise of arterial and fall of venous pressure (20″ to 30″) followed by (2) a gradual return to and *beyond* the previous state of heightened venous lowered arterial pressure (2 to 3 min.). This hyperdilatation is note-

worthy; it is greatest after most intense stimulation, and is to be regarded as an exhaustion phenomenon—there remained after section a tonic influence—where? in the peripheral centres, say D. and M. With the plantar nerve, digital vein and digital artery, they obtained identical results. Hence the conclusion that there is no more reason to suppose dilatators in the sciatic than in the sympathetic, since in both the effects of irritation are the same, viz., primarily contraction; secondarily, dilatation. Resuming now the general question of dilatation, viz., paresis of contraction, whence may the suspension of activity proceed? from spinal or from peripheral centres? In the above experiments dilatation follows the interruption of an activity above the section, the exhaustion of an activity below the section; on the other hand nothing proves that the paralytic dilatation is a fatigue, for it should then always be preceded by a period of contraction, but we must admit that it is a primary effect. The effects cannot here be explained as an inhibitory interference of spinal over peripheral centres, and dilatation can probably occur in either of two ways, viz. by paresis of spinal centres or by paresis of peripheral centres.

Etude sur la Physiologie des Nerfs des Muscles Striés. S. Tschiriew. (*Arch. de Phys.*, 1879, Nos. 3 and 4.—In a former paper (Tonus quergestreifter Muskeln, Arch. f. Anat. u. Phys., Phys. Abth. 1879, p. 89.)—T. pleads strongly in favour of the existence and reflex mechanism of muscular tonicity. The present paper continues to consider the theory of reflex muscular action by soliciting impression, and deals therefore with the nature, peripheral source, channels and presiding centre of the activity. Firstly, with regard to common sensibility, he remarks that normally muscle is insensible to pain by an instantaneous irritation, but sensitive to prolonged irritation, and also in certain pathological states (myalgia rheumatica, cramp, psoïtis, hysterical muscular hyperæsthesia, amyotrophic lateral sclerosis), and that the phenomenon of pain necessarily entails centripetal nerve-fibres. How do we judge of weight, or of the attitude and movement of our limbs? By some a specific sense of muscular tension ("the muscular sense") is imagined to arise in the contracting fibre; by others the sense of effort is attributed to the motor cell, being the subjective aspect and measure of its intensity of action; and by others still "muscular sense" is declared to be an entire misnomer, sensation being informed by altered tension, not of the muscle but of surrounding tissues. T. declares in favour of the peripheral

origin of a centripetal muscular sense by virtue of a special sensibility. Passing to the question of reflex movements by excitation of muscles, T. discusses the knee-phenomenon, and condemns the old theory of direct excitation of muscular fibre by mechanical tension; he refers it to a dragging upon the aponeurotic nerves by the percussion of the already stretched tendon, and asserts that the reflex is effected in that part of the cord whence the crural nerves originate—he estimates the interval between percussion and contraction at $\frac{1}{30}''$. His general conclusion is that muscles are connected with the cord by motor nerves and also by centripetal nerves, viz., by a nervous arc.

In a previous paper ('Sur les Terminaisons Nerveuses dans les Muscles striés,' Archives, 1879, p. 89) he claims to have proved that on striated fibres there are none but motor terminations, and that non-medullated nerves of muscle ending by arborisation in the aponeuroses are those of muscular sensibility.

Having regard to cases where there is "delay of pain," T. states the opinion that all sensations common and special are conveyed to the cord by common centripetal fibres, but are dissociated into separate channels by the agency of the cells of the grey matter. He anticipates, therefore, that delay of pain may be found associated with lesions limited to the posterior horns in correspondence with the affected district, and conversely that impairment of sensation without delay of pain should rule when lesions are confined to the posterior roots or columns.

T.'s paper concludes with a criticism of Krause's "hypothesis of discharge," a hypothesis based on a fallacious comparison of the motor end-plate with the plate of the electric organ. His previous paper ('Sur les Terminaisons,' &c.) went to prove that the terminal arborisation of the axis-cylinder, is the essential element of an end-plate, and not the granular layer. He adheres to Du Bois-Reymond's "modified hypothesis," advancing experiments to show how the shape of the end-plate should favour the concentration of the excitor influence of a disturbed electric equilibrium.

Dr. Gowers (*Med. Chir. Trans.*, vol. lxii. p. 269) estimates the mean "patellar tendon reflex" interval at $\frac{1}{10}''$ (·09 to ·15), and remarks that this corresponds with the *à priori* time value assignable to the reflex act (viz., ·045″ for conduction, ·05″ for central action, ·01″ for latent muscular period). He calls attention to a small rise in his tracings, about ·05″ after percussion, and asks whether this be not the indication of a *direct* muscular contraction by the *direct* irritation of muscular tension, the subsequent wave

indicating the indirect contraction by the *reflex* excitation of tension. G.'s account of the knee-phenomenon would thus include both its leading theories, viz., that of direct irritation and that of reflex excitation. For the front-tap contraction (a name given by G. to the gastrocnemius contraction, following a sharp tap on the anterior leg muscles), he finds an interval of ·04 to ·05, and concludes that the contraction is one of *direct* irritation. The ankle-clonus is regarded by G. as due to the direct stimulation of a muscular irritability, constituted by the reflex effect of tension, its frequency being 6 to 8 per sec., while the rare knee-clonus is regarded as reflex in mechanism, its frequency being 2·5 per sec. He considers clonus as an instance of a rhythmic action effected by a continuous stimulation.

NOTE.—In the course of measurements, which will be detailed elsewhere, of the interval between percussion and contraction, I found for the knee phenomenon ·04″ as its mean value in normal subjects (Gowers, ·09″ to ·15″; Tschiriew, ·033″). For the front-tap interval I found ·035″, ·04″, and ·04″ in three cases where the knee interval was measured to be ·035″, ·04″, and ·045″ (Gowers, ·04″ to ·05″). In two other cases (paraplegia) I found for the ankle-clonus a spasm frequency of 8 and 9 per sec., and for the knee-clonus of 9 and 10 per sec.

I may remark that a knee clonus does not seem to be so rare as it is usually stated to be. *A priori* we should not expect a rectus-clonus to be more unusual than a gastrocnemius-clonus, and it is simply owing to the respective circumstances of the two joints, that the spasms can commonly be elicited single in the rectus, multiple in the gastrocnemius. In point of fact a knee-clonus may be elicited by appropriate measures even in normal subjects, which is to the pathological phenomenon what the foot-trepidation of a normal subject is to its exaggeration—ankle-clonus. I have found the knee-clonus in the ankle-clonus cases I have examined by a flexing jerk of the rigidly-extended limb (best in the rigidity of paraplegics, where there remained some degree of voluntary motility).

The identity in the time-intervals of the single spasms at the two joints argues them to be of identical mechanism; the identity in the spasm-frequencies argues these also to be of identical mechanism. By other considerations that cannot be entered upon here, I am led to the conclusion that *all* the spasms above referred to are reflex from the spinal cord.

A. WALLER.

Primary Athetosis. GNAUCK. (*Arch. f. Psych.*, ix. p. 300.) —A girl, aged 13, who had always been healthy and had no predisposition to nervous disease, was seized with smarting pains in the right face. In eight days, continuous involuntary movements of the right hand and foot were observed, and shortly afterwards slight drooping of the right side of the face. Treatment with bromide of potassium was followed by disappearance first of the facial pains and then of the involuntary movements, these last

being completely gone in three months. The drooping of the face became less noticeable, but did not disappear. She remained well about three months, when her old symptoms reappeared. She noticed now that in certain places her sense of touch was not as acute on the right as on the left side. The facial pains ceased after an attack of epistaxis, but the other symptoms persisted. In this condition she consulted the author, who noted movements in the fingers, hand, forearm, toes, foot, and leg of the right side. The movements were slow, rhythmical, unceasing, involuntary, and yet, as it were, purposive, and they ceased during sleep. There was facial hypokinesis on the right side. The cutaneous sensibility was somewhat diminished in the parts in which the movements occurred. But there was no loss of power in the right extremities; the electric irritability was unchanged; the reflex excitability, including the phenomenon of tendon-reflex, was normal, and the circumference of the limbs was the same on both sides. Bromide of potassium was given in increasing doses, and the continuous current, from the cervical and lumbar regions of the cord downwards to the affected muscles, was applied every other day for about ten minutes. In a few months the patient had completely recovered.

The important points in this case are (1) the fact that the disease occurred in an individual who had previously enjoyed good health; (2) the partial recovery and subsequent relapse; and (3) the ultimate complete recovery of the patient. The case is one of idiopathic or primary athetosis, as distinguished from secondary or symptomatic athetosis, in which we have a history of hemiplegia, epilepsy, brain atrophy, &c. Of the primary disease only five cases are on record, and Gnauck gives a comparative analysis of them. In the only instance in which an autopsy was made, a focus of softening was found in the 'corpus striatum and lenticular nucleus' of one side. In the case just reported there was probably an affection of the outer part of the left half of the pons. The author distinguishes between symptomatic athetosis and cases of athetoid movements: in the former, the symptoms very closely resemble those of primary athetosis, in the latter, they differ from these in some important respects.

W. J. Dodds, M.D., D.Sc.

A Case confirming Cerebral Localisation. Dr. Tamburini (*Rivista Sperimentale di Freniatria, Anno V. Fascicolo III.*) gives an account of an imbecile, 45 years old, bearing the honoured name of Paul Veronese. He was subject to epileptic fits, which were often

followed by delirium. During an attack of this kind he killed an idiot with a knife. The epilepsy dated from infancy, and was accompanied by atrophy and paralysis of the left arm and leg. He was four years and a half in the asylum at Reggio, where he died. The fits had become so frequent that they could not be counted. The attacks were always preceded by an aura; then followed clonic movements, which began in the paralysed and wasted left arm. Sometimes the convulsions were limited to the arm, and extended to the face and leg of left side; sometimes they involved the whole body. The face was less developed on the left side than on the right. In fact the whole left side was smaller than the right; still the leg was not so much paralysed as the arm. The man's intelligence was very deficient, the power of speech limited, and the command of the vocal apparatus imperfect. Words were pronounced with great effort and with repetition and stuttering.

The left hemisphere of the brain was found to be normal, it weighed 510 grammes, but the right hemisphere was much smaller, weighing only 260 grammes; the whole brain weighed 930. On the right side, the ascending parietal convolution (the posterior median of Ecker) was atrophied and indurated, especially at its external part. This atrophy and induration involved the neighbouring portion of the ascending frontal gyrus anterior median of Ecker and the lower parts of the second and third frontal gyri. Behind this circle of sclerosed matter there was a cavity about the size of a pigeon's egg, full of purulent matter, extending through the left temporal lobe and occupying the place of the island of Reil, all vestiges of which had disappeared. The right thalamus opticus, crus cerebri, and surface of the pons on the right side were also found diminished in size and hardened in texture. The spinal cord was found atrophied, especially on the cervico-dorsal region. The sclerosed part examined by microscope showed an increase in the connective tissues and in the gyri round the fissure of Sylvius; amyloid corpuscles and white cells were found around the vessels.

Tamburini remarks that the atrophy of the left hemisphere continued into the crus cerebri, the pons and anterior pyramid of the same side, and then passing into the half of the spinal cord taken into connection with the atrophy and paralysis of the left side of the body, furnishes a striking proof of the dependence of the trophic and motor functions, not only in the cord and pons, but also in the cerebral hemisphere as well as of the crossing of the nervous tract denied by Brown-Séquard.

The loss of power in the left arm and leg and in the face is explained by the injury to the external portions of the ascending parietal, the ascending frontal, and the lower part of the third frontal.

The dissection of the brain, writes the learned physiologist, signally confirms the criterion for diagnosis brought to light by Hughlings-Jackson and experimentally confirmed by us (Tamburini and Luciani) that the lesion of the motor centre of the cortex cerebri may be diagnosed from the muscles first affected by the epileptic attack. The injury to speech is explained by the destruction of the island of Reil, which, in this case be it noted, was on the right side of the brain.

In considering the atrophy and sclerosis of the right optic thalamus, Tamburini enumerates the views of some distinguished neurologists on the function of this large basal ganglion. Luys holds that it contains four special centres for the representation of smell, sight, hearing and general sensibility. Ferrier thinks it to be a ganglion of interruption or centre of convergence of the sensory tracts. Fournier, Carville, Duret and Crichton-Browne hold that it is a centre of general sensibility. Nothnagel found that destruction of the optic thalamus was followed by a loss of sensibility and also of voluntary motion. Meynert, on the other hand, holds that the sensory impressions coming from the periphery are transformed in the optic thalami into movements; they are thus the automatic centres of reflex unconscious motions. Schiff, Lussana and Lemoigne concluded from their experiments that the optic thalami are centres for the motions of the arm and hand of the opposite side. The fact that in this case the atrophy of the optic thalamus was a continuation of the affection of the motor gyri, induces Dr. Tamburini to think that the thalamus is a conductor of the motor fibres, especially of the upper limb.

Hardening of the hippocampus major was found in this case; analysing the observations of five pathologists, he shows that it was found in 60 epileptics out of 272 dissections. He is of opinion that this lesion is only found in old cases of epilepsy. According to Meynert, the cornu ammonis contains peculiar pyramidal cells, but the destruction of the hippocampus causes no loss of any motor powers, nor does its removal bring any modification in epileptic convulsions. Tamburini recalls the views of Ferrier, who places the seat of olfactory sensation in the subiculum of the hippocampus. For these reasons he concludes that the hardening of

this portion of brain has no causal influence on the production of epilepsy. He considers that the morbid process, so well and learnedly described, commenced in the cortex cerebri, and then descended through the basal ganglia to the spinal cord.

Caizergues' Case of Cerebral Localisation.—Dr. Caizergues, in a pamphlet of eleven pages, which first appeared in the 'Montpellier Médical,' describes a case of cerebral hæmorrhage, in which the new views as to the functions of the cortex cerebri and white matter of the centrum ovale are well illustrated. The man, 38 years of age, was admitted into the hospital at Montpellier with mitral contraction and insufficiency and contraction of the aorta. The arteries were believed to be atheromatous. Suffering from diarrhœa he rose from his bed, and returned exclaiming, "I cannot move my fist." Then a feeling of tingling, similar to what he had felt in the arm, appeared in the right leg, and ten minutes later the whole right side was paralysed. He was heard to say to the religieuse, "Ma mère," and these were the last intelligible words he was heard to speak, though he tried to make himself understood by signs. The interne ascertained the presence of hemianæsthesia nicely limited to the right side of the body. There was no trembling or convulsions. He died about three hours after the first attack. An effusion of blood was found in the left hemisphere, which stretched in an oblique direction from above downwards and from behind forwards. The blood had raised the convolutions of the motor zone without injuring their tissue, and had gained the surface at one point near the union of the middle and upper third of the ascending parietal (posterior median of Ecker). The mass of the clot, about the size of a small apple, extended obliquely from below the fissure of Rolando to the fissure of Sylvius. Neither the nucleus lenticularis nor the nucleus caudatus was touched, but the internal capsule which separates them was found destroyed at its posterior and external part. The optic thalamus had escaped injury.

Dr. Caizergues thinks that the blood escaped gradually from the giving way of several miliary aneurisms. He holds that the paralysis of the arm and then of the leg was due to the destruction of the conducting white fibres of the centrum ovale, and not to any lesion of the motor area of grey matter, which Dr. Caizergues regards as left uninjured, although the blood forced its way to the surface at one point. The hemianæsthesia is explained by the destruction of the internal capsule of the corpus striatum, and

the aphasia is explained by the rupture of the lower pediculo-frontal fibres.

William W. Ireland.

Lactic Acid as a Hypnotic.—Dr. Maragliano has recorded in the *Rivista Sperimentale di Freniatria* (*Anno V. Fascicolo III.*) the result of some experiments upon the hypnotic and sedative properties of lactic acid upon the insane. He mentions two theories upon the production of sleep. Some physiologists have advanced that under the influence of fatigue oxygen disappears from the blood, causing inactivity of the nervous centres and sleep, but the provision of oxygen being renewed, their diminished powers are restored, and the man awakes. Others hold that, though the quantity of oxygen in the blood is not less during sleep than during the waking state, it is quickly absorbed by those materials, such as creatine and lactic acid, which are the result of the disintegrating activity of the bodily organism, and which require to be oxidised ere they are eliminated. When this oxidisation has been accomplished, the brain can again make use of the oxygen in the blood and resume its proper functions.

According to the experiments of Ranke and Preyer, the artificial introduction of lactic acid and other products of retrograde metamorphosis are capable of producing weariness and sleep. With this theory in mind a number of German physicians used lactate of soda or lactic acid in the treatment of sleeplessness, but the results were somewhat disappointing. Doctors Maragliano and Sepilli tried these drugs in about a hundred cases of insanity. They found that if given (lactic acid in doses of 8 to 10 grammes and lactate of soda 12 to 15 grammes) three or four hours before bedtime, they were sufficient to subdue the insomnia of quiet melancholia; but they had little or no effect if given immediately before going to bed. In more decided cases of agitation and sleeplessness they were found very inferior to chloral and morphia, besides being too costly for ordinary use. They were also apt to produce nausea and vomiting.

W. W. Ireland.

Relations of Brain, Mind, and Higher Nerve Function.—In an able address delivered at the beginning of the session of the Pathological and Clinical Society of Glasgow, Dr. Alexander Robertson discusses some of the pathological and physiological relations of brain, mind, and higher nerve function. He begins by

giving a short sketch of the simpler forms of nervous apparatus in the lower animals, then proceeds to consider at length the theory of localisation of brain function, and particularly as it relates to the cerebral convolutions, "how far it seems worthy of acceptance, and how far it seems problematical." An epitome of physiological, pathological, and anatomical evidence in support of the theory of localisation is then gone over, from which it may be gathered that the greatest amount and most important part of the evidence is gained from a study of pathology, and more especially from the consideration of such diseases as aphasia, paralysis, and Jacksonian epilepsy.

Next in importance is the physiological evidence, in which some of the results obtained by Ferrier and Hitzig by electrical stimulation are discussed. Dr. Robertson says that of course there is no doubt about the facts elicited by these observers. It is in the interpretation of these facts that the difficulty lies; that is, whether the purposive movements, and so forth, brought about by stimulation of the cortex, are simply due to conduction of electrical currents from the grey matter of the surface to the basal motor ganglia; or depend on mere conduction of electricity from the motor centre to the basal ganglia by means of special fibres for the particular movements induced; or are due to reflex action, the surface in the area of the apparent motor centres being centripetal or afferent to the corpus striatum; or again are due, as Brown-Séquard proposes, to inhibition; or, lastly, as Hitzig himself advocates, to "what may be called a sensory explanation" of the apparent motor centres.

The anatomical evidence referred to by Dr. Robertson does not call for special reference, but he is of opinion that on the whole it leans to the theory of localisation. Dr. Robertson goes on to say that in the light of cortical localisation we arrive at a more satisfactory explanation of many forms of paralysis, and are able to account better for the grouping of symptoms and general course of some other forms of nervous disease. Take, says he, the case of general paralysis of the insane. In this disorder psychical disturbance usually precedes defective articulation, but occasionally they begin simultaneously. Now the presence of motor centres in the superficial grey matter affords a ready solution of the difficulty, showing how there may be associated two kinds of symptoms apparently so different from each other, as delusion and defective articulation, but really so closely allied since the motor intuitions of language are so deeply concerned in the exercise of thought. And it is

worthy of note that the adhesion of the pia mater to the convolutions is most marked in the frontal and parietal regions, a fact which supports the theory of localisation so far as it applies to motor centres.

The hypothesis of the psychical and motor functions being in such close relation also goes far to account for the frequent manifestation of motor symptoms in other forms of insanity.

W. G. Thompson, M.D.

Traumatic Insanity.—Dr. Brower (*Chicago Medical Journal and Examiner*, December 1879) believes that the production of insanity by injury to the head probably depends less on the nature of the injury and its associated phenomena than on the inherent peculiarities of the individual. Fatal injury to the brain may result from sudden violence inflicted on the skull without injury to the cranial bones. Dr. Brower had under observation during the late rebellion "a case of gunshot-wound of the head, that in twenty days resulted in death, and post-mortem examination showed no injury to the cranial bones, but an abscess in the brain and associated meningitis and encephalitis. The case was one, doubtless, of contusion of the brain with solution of continuity of its constituent elements, and without injury to the bone, the skull undergoing change of form suddenly, and the cerebral substance offering less resistance than its bony covering, extravasation and laceration of brain-tissue ensued." This author believes that if the connection between the injury and a criminal act is complete, "the history will show a neurophatic diathesis—will show that after the injury cephalegia was frequent, that the sleep was insufficient in quantity, or else disturbed by dreams; that after a time change was noticed in the emotional and then in the intellectual part of the mind; in rare cases both may occur simultaneously."

Bullet-wound of the Brain.—Dr. E. F. Brodie records (*American Practitioner*, January 1880) an interesting case of this form of injury. A man, aged 22, received on August 11, 1879, a wound of the cranium from a pistol-ball. The ball, which weighed half-an-ounce, entered at the supra-orbital foramen, fractured the orbital roof and temporal fossa, passed beneath the integument to a point two inches and a fourth above the meatus auditorius externus and out, dividing the posterior branch of the temporal artery. Several spiculæ of bone were removed, and about half-an-ounce of

cerebral tissue issued from the point of entrance. The patient was semi-comatose for about five days. On the tenth day he, having been kept on low diet, became so excessively hungry that his friends imprudently allowed him to eat bread and bacon, smoke a cigar, "and enjoy a convivial round generally," during which his outraged system revolted. He had hæmorrhage from the posterior branch of the temporal artery to the extent of five quarts. The wounds suppurated, and four days later a scale of lead, weighing 28 grains, was removed from the roof of the orbit, and also several large spiculæ of bone. In six weeks the wounds closed, leaving no symptoms beyond slight impairment of memory and stiffening of the muscles of the jaw. Subsequent observation of this case would be interesting and instructive.

ROBERT LAWSON, M.B.

// BRAIN.

JULY, 1880.

Original Articles.

ON THE FORM AND TOPOGRAPHICAL RELATIONS OF THE CORPUS STRIATUM.

BY PROFESSOR J. C. DALTON, NEW YORK.

In the annals of the Anatomical and Surgical Society of Brooklyn, New York, for January, 1880, I gave a résumé of the main features of structure of the brain, as now generally understood by cerebral anatomists, together with a special description of the corpus striatum. These two subjects have a necessary connection with each other, since the corpus striatum is found, on examination, to reproduce very nearly the general curvilinear arrangement of the cerebral convolutions. The description here given of its special configuration is mainly in the form of a transcript from my former article.

If we leave out of consideration for the present the cerebellum and some of the smaller collections of grey matter in the cerebrum, the structure of the cerebro-spinal axis, according to the ideas now in vogue, may be simply described as follows:—The grey matter of the spinal cord, surrounding its central canal, is continued upward on the floor of the fourth ventricle and around the aqueduct of Sylvius. This continuous layer is the "grey matter of the medullary canal," and from it all the cerebro-spinal nerves of motion and general sensibility take their origin. It is connected by longitudinal tracts of white substance with the parts above; and these

longitudinal tracts, under the name of the crura cerebri, reach a second division or deposit of grey matter at the base of the brain, known as the corpus striatum and optic thalamus, or the "cerebral ganglia." The ascending fibres of white substance pass through and between these ganglia in the form of a fan-shaped layer, the "internal capsule;" and from its upper border they emerge into the diverging expansion of the corona radiata, to reach finally the third and largest mass of grey matter, which is spread out in the convolutions of the cortex.

In this diagrammatic schedule of the structure of the encephalon many things of course are omitted; such as the transverse commissures, connecting similar parts of the two hemispheres, and the antero-posterior tracts, connecting different parts of the same hemisphere. The question whether some fibres of the crus cerebri and internal capsule may not be continuous, from the spinal cord to the cerebral convolutions, is also left untouched. But for our present purpose the above account will represent sufficiently well the relations of the white and grey matter in the cerebro-spinal system.

The spinal nerves and nerve-roots connect the peripheral organs with the grey matter of the medullary canal. The columns of the cord and the crura cerebri connect this nervous centre with the cerebral ganglia; and from the cerebral ganglia the fibres of the internal capsule and corona radiata are the organs of transmission to the cerebral convolutions. Thus the intermediate station or deposit of grey matter, between the cerebral convolutions above and the spinal cord below, consists of the corpus striatum and the optic thalamus.

Of these two bodies the optic thalamus is the more homogeneous in appearance, since the white fibres which penetrate it rapidly separate from each other, giving to the ganglion a comparatively uniform light grey tint. The whole of the thalamus, furthermore, lies on the median side of the internal capsule. On the other hand, in the corpus striatum the fibres run, for the most part, in distinct bundles to within a short distance of its outer border, giving it the visibly striated appearance to which it owes its name. Moreover, this ganglion is split almost completely into two portions by the internal capsule, which lies between them. As the internal

capsule is directed obliquely from below upward and from within outward, one portion of the ganglion lies inside and above it, the other outside and below it. The former is the *intraventricular* portion, or that which is visible from the cavity of the lateral ventricle. It has a club-shaped extremity, or head, directed forward, and a slender continuation, or tail, directed backward; whence its name of the "caudate"

Fig. 1.—Diagram of Brain in Transverse Vertical Section.

1. Crus cerebri.
2. Internal capsule.
3. Optic thalamus.
4. Corpus striatum.
5. Surcingle.
L. N. Lenticular nucleus.
S. Fissure of Sylvius.
Fo. Gyrus fornicatus.

F'. First frontal convolution.
F''. Second frontal convolution.
F'''. Third frontal convolution.
T'. First temporal convolution.
T''. Second temporal convolution.
T'''. Third temporal convolution.
H. Gyrus hippocampi.

nucleus. The other portion, not being visible from the ventricle, but covered by the internal capsule and imbedded in the substance of the brain, is known as the *extraventricular* portion; or, from its lens-like figure, the "lenticular" nucleus. It is now customary to confine the term "corpus striatum" to the first or intraventricular portion of the ganglion. This is

the part to which my observations especially refer, and which I shall describe under the name of corpus striatum. The remaining portion is designated only as the "lenticular nucleus."

The relations of the parts above enumerated may be shown by a vertical and transverse section of the brain, as in Fig. 1. This figure is a diagrammatic one, but it indicates the general direction of the fibres of the crus cerebri, the internal capsule, and the corona radiata. It is also seen that both the optic thalamus and the corpus striatum are above and on the inner side of the internal capsule, while the lenticular nucleus is outside and below it. This nucleus, as is well-known, is divided by thin laminæ of white substance into three nearly concentric layers or zones.

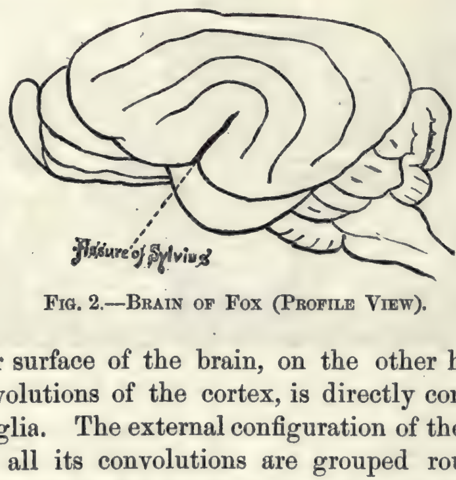

FIG. 2.—BRAIN OF FOX (PROFILE VIEW).

The outer surface of the brain, on the other hand, formed by the convolutions of the cortex, is directly connected with central ganglia. The external configuration of the hemisphere shows that all its convolutions are grouped round a point corresponding with the internal capsule and the lenticular nucleus. This is most distinctly visible in such a brain as that of the fox (Fig. 2), which is marked only by a few simple and parallel convolutions. The constant and deeply marked fissure of Sylvius runs upward and backward, forming the line of division between the frontal and temporal lobes; and all the convolutions on the convexity of the hemisphere are curved very regularly around this fissure. They run upward and backward in front of it, encircle its upper extremity, and then return downward and forward to the end of the temporal

lobe. The entire brain has the appearance of being folded, with all its convolutions, about a transverse axis passing through the bottom of the fissure of Sylvius.

In the human brain, Fig. 3, the fissure of Sylvius is equally distinct, and it is easy to see the same general arrangement of the convolutions running round its upper extremity. The three frontal convolutions pass upward and backward on the convexity of the hemisphere, and the three temporal convolutions run downward and forward, as in the fox, beneath the

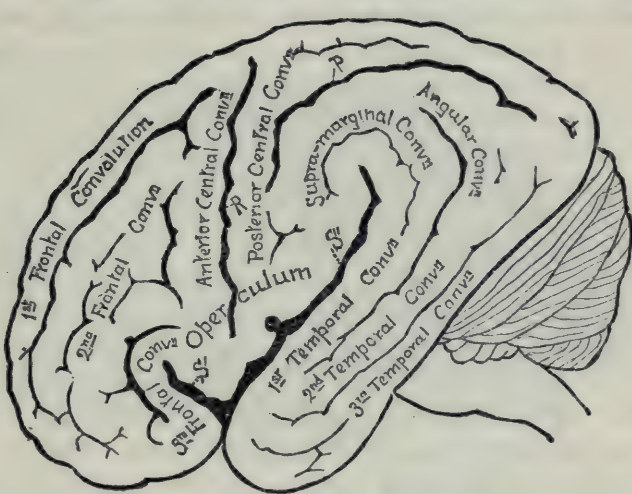

FIG. 3.—HUMAN BRAIN (PROFILE VIEW).

S'. Fissure of Sylvius, anterior branch. R. Fissure of Rolando.
S''. Fissure of Sylvius, posterior branch. P. Interparietal fissure.

fissure of Sylvius. But in the human brain, as compared with that of the animal, there are two new features. In the first place, the fissure of Sylvius is double. There is an anterior branch and a posterior branch, and included between them is a triangular-shaped portion known as the "operculum." Secondly, in addition to the fissure of Sylvius, and above it in position, there is another deep fissure, running in a nearly vertical direction from above downward, on the side of the hemisphere. This is the fissure of Rolando. It is accompanied by two convolutions having a similar direction, one in front and one behind, the "anterior and posterior central

convolutions." The fissure, with its accompanying convolutions, seems to cut across the general direction of the curvature nearly at right angles, and consequently to introduce a certain degree of confusion into the plan of the cerebral hemisphere. But this is only an appearance. It is due simply to the greater development, in man, of the cortical substance.

At the bottom of the Sylvian fissure, the side of the brain is occupied by the convolutions of the *insula*, or "island of Reil." The insula corresponds in position with the outer surface of the lenticular nucleus, and it is that part of the cortex of the brain which is in most immediate proximity to the cerebral ganglia. Externally it is concealed from view, because that portion of the cerebral hemisphere included between the two branches of the Sylvian fissure projects downward from above, and overhangs it like a cover. It is for that reason this portion is named the "operculum;" and when the two branches of the Sylvian fissure are opened, and the operculum between them lifted up, the insula, with its convolutions, is exposed at the bottom of the cavity.

It is evident, therefore, that the fissure of Rolando is not really an interruption to the general run of the convolutions. In the fox the fissure of Sylvius is single, and the convolutions are folded upon themselves only once. But in man the fissure of Sylvius is double, and the convolutions are folded upon themselves twice. The development of the cortex in the human brain is so great that different parts of it overlap each other. The operculum hangs down over the insula. The longitudinal convolutions, instead of running in a continuous curve from before backward, make, about the middle of their course, an additional crook. They turn downward along the anterior branch of the Sylvian fissure, and again upward along its posterior branch. In that way they form the operculum. The anterior and posterior central convolutions are the parts immediately folded upon each other in this downward angular bend; and the dividing line between them is the fissure of Rolando.

The median surface of the hemisphere, Fig. 4, shows a very similar arrangement of its convolutions; the central one, known as the "gyrus fornicatus," being continued, in an

arched or vaulted form, around the end of the crus cerebri, the internal capsule, and the cerebral ganglia. It starts from the inferior part of the anterior lobe, just in front of the Sylvian fissure. It turns up round the anterior extremity of the corpus callosum, and runs along its upper border. Here it is included between the corpus callosum below and the fissura calloso-marginalis above. It extends backward, in this part of its course, as a single independent convolution, as far as the ascending branch of the fissura calloso-marginalis. There it makes one or two connections with the præcuneus,

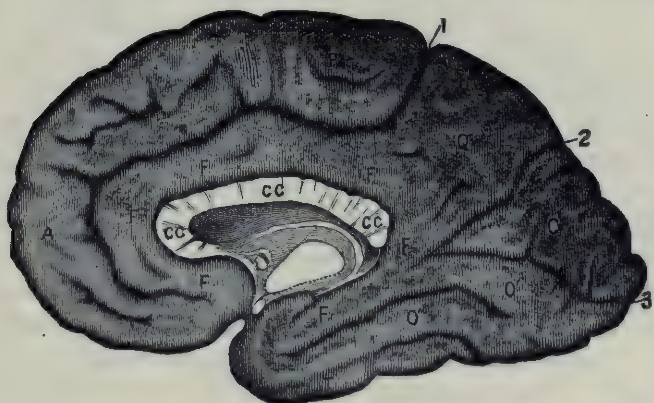

FIG. 4.—HUMAN BRAIN; MEDIAN SURFACE OF THE RIGHT HEMISPHERE.

A. First frontal convolution.
F. Gyrus fornicatus.
P. Paracentral lobule.
Q. Lobulus quadratus, or Præcuneus.
C. Cuneus.
O′, O″. Occipito-temporal convolutions.
T. Third temporal convolution.
c. c. Corpus callosum.
1. Fissura calloso-marginalis.
2. Fissura parieto-occipitalis.
3. Fissura calcarina.

and farther back with the cuneus also. It then curves round the posterior extremity of the corpus callosum, passes beneath the crus cerebri, and runs downward and forward to a point just behind the fissure of Sylvius, almost exactly opposite the place from which it started. In this lower portion of its course it is often designated as the "gyrus hippocampi;" and it in fact forms the hippocampus major by the inflection of its grey matter in the inferior horn of the lateral ventricle.

The final destination, therefore, of the fibres of the internal capsule and the corona radiata is the grey matter of the

cortex. That part of the cortex nearest the cerebral ganglia is the insula; and all the rest of the cerebral convolutions are arranged, in a more or less continuous curve, around this spot. All these points are tolerably well known, and receive the general assent of anatomists who have paid particular attention to the brain.

But there are some features in the form and topographical relations of the corpus striatum which are not so well understood. By the term corpus striatum, as already mentioned, we now designate only the intraventricular nucleus of the ganglion, or that which lies above and on the inside of the internal capsule. It is usually described as consisting of a thick or club-shaped portion directed forward, and a slender tail-like extremity directed backward, which runs along the outer edge of the lateral ventricle, and terminates somewhere about the posterior border of the optic thalamus. In reality it is much more extensive than this. A few writers speak of it as reaching into or to the inferior horn of the ventricle; but the only ones, so far as I know, who have described it in a really satisfactory way are Gratiolet, Todd, and Ludovic Hirschfeld. The best account of all is that given by Gratiolet, in his 'Anatomie comparée du Système nerveux,' published in 1857. This description is generally ignored by present writers, and it is expressly stated by so high an authority as Henle, in his last edition, 1879, that the tail-like process of the corpus striatum "tapers gradually from before backward, and terminates in a point opposite the end of the optic thalamus." ('Handbuch der Nervenlehre,' Braunschweig, 1879, p. 155.)

On the contrary, the real form of the corpus striatum is almost that of a complete ring. This can often be seen quite distinctly, without further preparation, when the lateral ventricle has been opened completely from the inside, as in Fig. 5. The slender continuation of the corpus striatum may then be traced, throughout its course, by the pinkish-grey colour of its substance, showing through the transparent lining of the ventricle. The body of the corpus striatum grows smaller as it extends backward in the upper part of the ventricle, and soon runs into the well-known tail-like pro-

longation. This prolongation reaches the posterior end of the optic thalamus, curves downward into the inferior horn of the ventricle, and then runs forward again to the anterior extremity of the inferior horn. It thus encircles the crus cerebri and internal capsule with a narrow band of grey matter, like a loop or surcingle; it ends below in the inferior horn of the ventricle, almost exactly opposite the point where it started in the anterior horn, and it repeats, within the cavity of the ventricle, the same folded or curvilinear arrange-

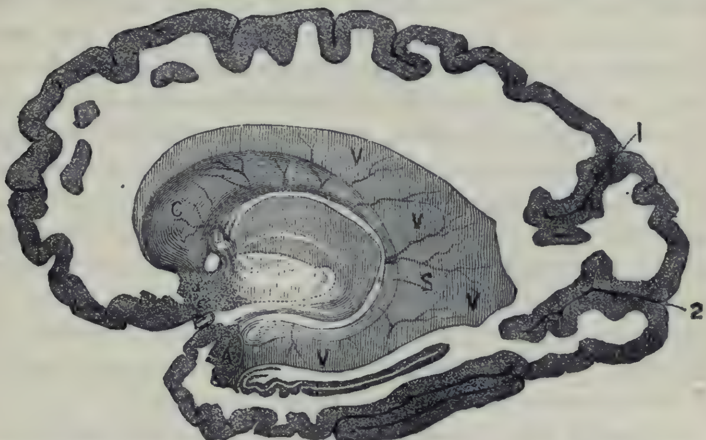

FIG. 5.—LONGITUDINAL AND VERTICAL SECTION OF THE RIGHT HEMISPHERE, SHOWING CAVITY OF LATERAL VENTRICLE (SEEN FROM THE INNER SIDE).

C. Corpus striatum. S. Surcingle. V. Ventricle. A. Amygdala. 1. Fissura parieto-occipitalis. 2. Fissura calcarina.

ment that is to be seen in the gyrus fornicatus and in the convolutions of the cortex generally.

The head of the corpus striatum is not only connected with the lenticular nucleus, but also with the grey matter at the base of the brain, just in front of the Sylvian fissure. The extremity of the surcingle, on the other hand, is connected with a deposit of grey matter forming the anterior wall of the inferior horn of the ventricle. This grey mass is known as the "amygdala." It is sometimes spoken of as an independent nucleus, but it is really continuous with the convolutions of the base of the brain, immediately behind the fissure of Sylvius.

In a view of the ventricle opened from the inner side it is seen that the curved prolongation, or "surcingle," of the corpus striatum is not of the same size throughout. It diminishes considerably in thickness while approaching the end of the optic thalamus, then increases somewhat during its downward curvature, again becomes more slender, and finally terminates at the front part of the inferior horn, in a visibly enlarged extremity. Very often, but not always, it is more or less interrupted near this enlarged portion by one or two bundles of white fibres, which cross it obliquely from above downward. These fibres come from the lower portion of the tænia semicircularis. The tænia semicircularis is well known, in the upper part of the ventricular cavity, as a slender ribbon of white substance, running from before backward along the floor of the ventricle, between the optic thalamus on the inside, and the corpus striatum on the outside. It accompanies everywhere the concave border of the surcingle, following the same curvature downward into the inferior horn of the ventricle, then runs forward along the roof of the inferior horn to its anterior end, and there terminates in the amygdala. It is a little before this termination that some of its fibres are seen to cross the grey matter of the surcingle.

The reason why this annular form of the corpus striatum is not more easily recognized in brain sections is that its two parts are so distant from each other and so variable in size. If a vertical and transverse section of the brain be made somewhat in front of the anterior commissure, it will show the head of the corpus striatum, in that situation, of very large size. In a similar section, passing behind the anterior commissure and through the front part of the optic thalamus, the corpus striatum will appear smaller; and in one passing through the middle of the optic thalamus it will be smaller still. It there begins to present the cut surface, not of the head of the corpus striatum, but of its tail-like appendage. If we continue to make sections farther and farther back, the appendage will at last disappear, and this disappearance will happen about the time the sections pass the posterior end of the optic thalamus. It is this fact, no doubt, which has led to the belief that the corpus striatum comes to an end about the same region. But

in reality it has not done so. It has curved downward in the space left between two successive sections, and has entered the inferior horn of the ventricle, to run forward towards its termination in the amygdala. Sometimes a section will happen to strike exactly the plane of curvature of this part, and will then show a more or less elongated tract of grey matter, running from above downward, a little to the outside of the optic thalamus. In a transverse section of the brain, through the middle of the optic thalamus, the caudate portion of the corpus striatum is visible above the internal capsule and lenticular nucleus (Fig. 1, 4). But there is also a smaller isolated area of grey matter below the level of the lenticular nucleus, and near the outer part of the inferior horn of the ventricle (Fig. 1, 5). Now these two sections, one above the internal capsule, and one below it, are the same thing, or rather they are two different parts of the same thing. One of them is that part of the corpus striatum which occupies the floor of the lateral ventricle above, the other is that which runs along the roof of the inferior horn below. In every section behind this, both these two parts will be visible, until a point is reached a little beyond the optic thalamus, when they will both disappear together.

The same figure shows how other parts of the brain, which are arranged in a looped form, may appear twice in a vertical section. In the brain of the fox (Fig. 2) it is evident that a section made at right angles to the fissure of Sylvius would cut the frontal convolutions in front of this fissure, and also the temporal convolutions, in reverse order, behind it. In Fig. 1 the same thing appears in the human brain. The frontal convolutions above the Sylvian fissure correspond in number with the temporal convolutions below it. In the parietal region their direct continuity is interrupted by the downward curvature about the fissure of Rolando; but still it is perfectly evident that the first temporal convolution (T') is really the end of the third frontal (F'''), the second temporal (T'') of the second frontal (F''), and the third temporal (T''') of the first frontal (F'). The remaining convolution of the temporal lobe, the gyrus hippocampi (H), is the termination of the gyrus fornicatus (Fo). In Fig. 4 the curvature of this convolution

is shown throughout its length. It is plain that, in a vertical section passing through the middle portion of the brain, it would be cut twice; once above and once below the situation of the cerebral ganglia.

It is not very easy to make a longitudinal section of the brain which shall pass continuously through the whole of the ring, or loop, formed by the corpus striatum and the surcingle. All parts of the loop are not exactly in the same plane, either vertically or longitudinally. Its posterior portions are situated

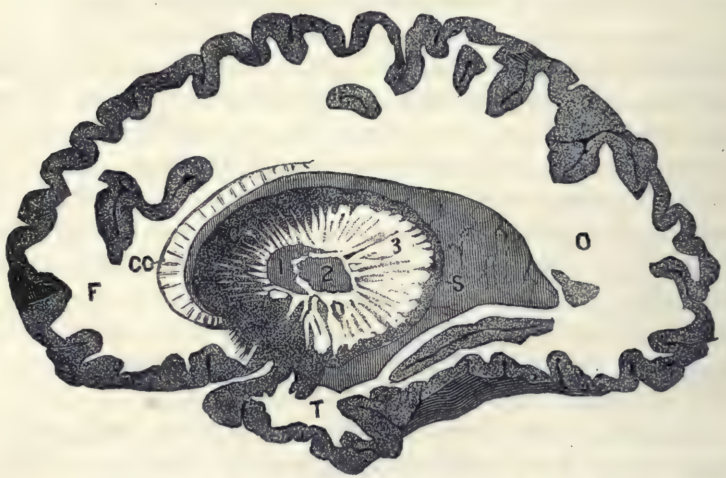

Fig. 6.—Longitudinal and Vertical Section of the Right Hemisphere, through Corpus Striatum and Surcingle (seen from the Inner Side).

F. Frontal lobe.
O. Occipital lobe.
T. Temporal lobe.
C. Corpus striatum.
S. Surcingle.
cc. Part of corpus callosum.
1, 2. Middle and internal zones of lenticular nucleus.
3. Diverging fibres of internal capsule.

farther from the median line than the anterior, and its superior and inferior branches are so slender and so widely separated that it is difficult to make a single oblique cut which shall show the whole of both. It can be done, however, by following carefully with the edge of a knife the curved track of grey substance from point to point, until the whole of it is exposed, and then removing the adjacent white substance down to the same level. Such a section is shown in Fig. 6.

It has exposed the white surface and radiating structure of

the internal capsule, and near its centre a little of the internal and middle zones of the lenticular nucleus. Surrounding the capsule is the grey tract of the corpus striatum and surcingle. In the head of the corpus striatum the bundles of white fibres are visible, traversing the grey matter in the form of distinct streaks, or striations, to within a short distance of its outer border. In the slender curved portion, on the other hand, no striations are to be seen, the colour being everywhere of a uniform grey. But throughout this region the radiating fibres of the internal capsule pass beneath the band of grey substance obliquely, from without outward, in the form of distinct bundles; and between these bundles the grey matter of the surcingle dips down, more or less deeply, at irregular intervals.

It is for this reason that transverse sections of this part of the corpus striatum vary so much in apparent size. If the cut happen to pass through a projecting bundle of the internal capsule, the section of the surcingle will appear small. If it pass in the interval between two bundles, the area of grey matter exposed will be considerably larger. From the same cause the sections are often unequal on the two sides of the brain. The projecting bundles of the internal capsule and the gaps between them seldom correspond exactly in the right and left hemispheres; and if they did, we could hardly make a section, either horizontal or transverse, which should follow with entire precision the same plane on the two sides. But, notwithstanding these apparent inequalities and variations, the grey matter is continuous throughout the curved portion of the corpus striatum.

In horizontal sections of the brain, the size and form of the cerebral ganglia vary greatly according to the level at which the section is made. At the level of the corpus callosum only a little of the arched portion of the corpus striatum is exposed, and none of the lenticular nucleus is visible. Somewhat below this, at the level of the fornix, the arched portion of the corpus striatum disappears, but there is a large oval section of its head in front, and a small one of a surcingle behind. Lower still the lenticular nucleus comes into view, separated from the head of the corpus striatum by the dis-

tinctly marked anterior prolongation of the internal capsule. At the level of the anterior commissure, as in Fig. 7, the

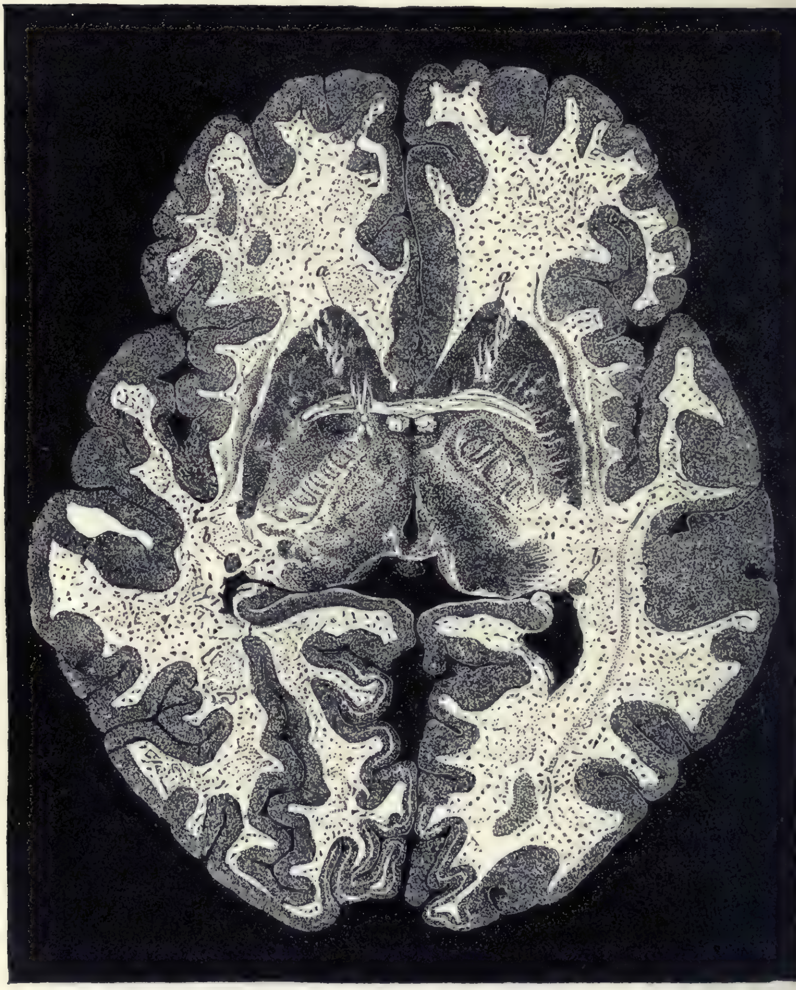

Fig. 7.—Horizontal Section of the Brain, through the Anterior Commissure and Lower Part of the Cerebral Ganglia (from a Photograph).

a, a. Head of corpus striatum. *b, b.* Surcingle.

lenticular nucleus and the head of the corpus striatum begin to fuse with each other, owing to the partial disappearance of the internal capsule between them. At this level the head of

the corpus striatum, *a, a,* is much reduced in size and altered in shape. The lenticular nucleus occupies a position outside and behind it; and still farther back, at *b, b,* is the section of the surcingle, which is here beginning to run downward and forward, along the roof of the inferior horn of the ventricle. At a lower section still, the head of the corpus striatum would be united with the cortical convolutions in front of the Sylvian fissure, and the surcingle with the grey substance of the amygdala.

NYSTAGMUS.

BY R. P. OGLESBY,

Ophthalmic and Aural Surgeon to the Leeds General Infirmary. Late Lecturer on Pathology in the Leeds School of Medicine.

NYSTAGMUS is the name of a disease arising from various causes of a distinct and separate character—difficult of generalisation—causes diametrically opposite—causes which on the one hand lead to grave results, on the other hand simple, but lasting in duration.

That it is a disease worthy of careful study is doubtless true, and if symptomatic cases of varied origin are met with, then it always repays the trouble of careful investigation.

I have primarily classed the disease under two heads—the idiopathic and the symptomatic, and afterwards redivided them into local and central.

The idiopathic form of nystagmus depends on a defective co-ordination of the associated movements of the eye, of a character essentially simple in form and not portending any grave result.

The oscillation of the eyeball varies in kind and degree. It may be horizontal (this is the most frequent form met with), or rotary, the eyeballs oscillating round the axis of the oblique muscles. The vertical form is seldom met with, the only case I have seen occurred in a man suffering from disease of the nervous centres.

It is recorded that binocular vision and nystagmus are incompatible, and truly so in the majority of cases; but where vision in either eye is but slightly impaired, the muscular apparatus not very unstable, the two eyes can act perfectly together during strong convergence.

The disease is peculiar to infant life consequent upon

various congenital abnormalities. It is also due in infant life to peculiar constitutional defects which early show themselves.

Idiopathic nystagmus is usually permanent, its causes being as a rule irremediable. It will be well to glance at the local form of the disease named congenital, and subdivide as follows:—

1st. *Albinoes.*—A very interesting form of the disease occurs in Albinoes. Marked examples of the kind were shown by me at a meeting of the West Riding Medico-Chirurgical Society. The subjects are two little boys—brothers—aged 6 and 8 years. Their hair is of a brilliant white; and their eyes, as they moved from member to member in the powerfully gaslighted room, oscillated horizontally with great rapidity. The family history disclosed physiological peculiarities concerning the child-bearing propensities of the mother. In these cases the unstable condition of the muscular apparatus is caused by absence of the hexagonal pigment cells of the choroid.

2nd. *Cataract.*—Lamellar or zonular cataract does not generally cause blindness. The marginal portion of the lens is sufficiently clear to allow rays of light to penetrate. Nystagmus usually accompanies this form of lens mischief.

3rd. *Choroiditis.*—Choroiditis is generally the result of inherited syphilis, and is by far the most prolific source of congenital mischief which causes nystagmus.

4th. *Anomalies of Refraction.*—Viz. myopia, hypermetropia, and astigmatism, are seldom accompanied by a nystagmus. Once or twice in myopes I have noticed this condition, but in these cases the ophthalmoscope disclosed an advanced disease of the choroid.

IDIOPATHIC NYSTAGMUS, ACQUIRED.

1st. *Opacities.*—On the surface of the cornea which implicate a large portion of the area of the pupil. In these cases it is necessary for vision that the eyes should perform rapid horizontal movements to bring the image rays to a focus on the retina.

2nd. *Sympathetic.*—A young man was admitted as an in-

patient under my care for treatment. His case, a somewhat novel one, presented peculiarities worth recording. In his youth he had lost the sight of the right eye from suppuration of the cornea, the result being a shrunken globe. Two years ago he sought the aid of a notorious quack who practises ophthalmology. (?) This man persuaded him to wear a glass eye over the diseased globe. When first seen by me he was still wearing the glass eye, which had produced a condition of sympathetic ophthalmia in the left eye. The pupil was partially closed, and vision limited to shadows. After dispensing with the glass eye, I removed the shrunken globe with great benefit, as vision in the left eye at once improved. In this case there was a very decided nystagmus of the rotatory kind.

With regard to the acquired form of idiopathic nystagmus, examples might with ease be multiplied, but the two cases given are sufficient to show how injury or disease will cause it.

The treatment varies much, and is at the best but of little good. In some cases tenotomy has been vaunted as a cure. I am afraid it is a proceeding of doubtful value. In such cases as are met with in Albinoes, coloured glasses often prove useful; and should the nystagmus depend on an anomaly of refraction —a thing very rare—properly selected glasses are of value.

The prognosis of idiopathic nystagmus is simple, and may be summed up in but few words. It is seldom dangerous and but rarely cured.

SYMPTOMATIC NYSTAGMUS.

Having glanced thus briefly at the idiopathic form of the disease, I must say a few words regarding a much more serious form, usually named Symptomatic Nystagmus. In the first form we have unstable muscular action, due to local causes, not interfering with the general health of the patient, and being more unpleasant than dangerous.

In the latter form we have a disease of central origin, often grave in its prognosis, and either due to unstable grey matter, such as that which will produce the petit mal, or a form much

more serious, as in myelitis or disseminated sclerosis. By far the greater proportion of cases depends upon a condition of irritation rather than direct structural lesion, and by far the greater proportion of cases lasts over a period indefinite in time, so far as clinical observation teaches us. In many instances the symptoms occur in early life and after an acute attack, often so severe as to endanger life, subside and remain passive for a time, afterwards to break out afresh, and so end in the patient's death. Again, nystagmus may be the early and most prominent symptom of undoubted intra-cranial mischief, of a kind that runs its course quickly and early destroys life. In these cases the patients are invariably young, and in many instances the lesion is due to acute inflammation of the base (basilar meningitis).

The etiology of nystagmus has many bearings. In some cases the actual nervous lesion is the sequela of acute febrile attacks. Typhoid fever is not unfrequently followed by serious visual ailments, chiefly—so far as my experience goes—by neuro-retinitis, and less frequently by nystagmus, the two representing a cerebral lesion consequent upon the acute meningitis during the fever. Acute rheumatism is another cause of the disease. Especially is it caused by those cases in which valvular disease of the heart is present. Severe and long-continued mental troubles associated, in the poor, with great privation and often absolute want of food. A case of this kind is at present under my care.

In one case the early cause was severe mental trouble concerning the payment of a large sum of money. The patient, who had a very sensitive and highly organised nervous system, completely broke down under the strain. Symptoms of disseminated sclerosis followed, nystagmus after some months appearing and becoming persistent.

The worst form of nystagmus I have as yet seen is persistent in a boy aged 10 years, and is congenital. The nervous system is ailing generally, and the whole symptoms very undoubtedly point to insular sclerosis, due, I believe, to inherited syphilis.

Pathology points to the following lesions as a cause of symptomatic nystagmus :—

1st. Lesions of sub-ventricular origin.
2nd. Lesions affecting medulla oblongata.
3rd. Lesions specially affecting grey matter of floor of fourth ventricle.
4th. Basilar meningitis.
5th. Miliary tubercle (meninges).
6th. Wounds of brain and medulla.
7th. Hæmorrhage (cerebral).

Epilepsy plays a marked part in the cause of symptomatic nystagmus, and in many cases the oscillation of the eyeballs is constant. This, so far as my experience teaches me, is never seen except in those who suffer from the minor form of the disease. In epileptics suffering from the graver form of the disease, the movements of the globes, although in character the same as in the minor form, are not persistent—commencing and ending with the fit.

A remarkable case of epilepsia gravior associated with nystagmus occurred in a boy aged 13 years, who was suddenly disabled by a sunstroke. When first seen by me as an infirmary patient, he was having as many as ten fits a a day. The aura was most distinct, commencing in the right arm and passing upwards along the neck towards the eye. This was followed by diplopia, and the seizure—rotatory nystagmus—always occurred during the paroxysm. Large doses of bromide of potassium, given thrice daily, after administration for a fortnight, produced a most beneficial effect on the character and the number of the fits. After taking the bromide for twelve months he reported himself as cured. This was not really so, for after the lapse of three months he again attended as an out-patient, saying that he had been quite free from the attacks till the week previous, since which time he had had three. He again attended for three weeks, and at the end of that period he assured me he was quite well, and I have not seen him since—now six months ago. In this case bromide of potassium, in 20-grain doses given three times a day, was the sole treatment, and the dose was never increased, neither was the drug given in combination with either digitalis or belladonna. Although rotatory nystagmus was most marked during

each paroxysm, the unstable condition was manifestly governed, so to speak, by the exhibition of the drug, and at last cured.

Another case of epilepsy associated with nystagmus equally interesting is worth recording. During the year 1873, I admitted as an in-patient a delicate, strumous-looking boy, who complained of failing sight. He had horizontal nystagmus, and the ophthalmoscope disclosed hyperæmia of the discs with venous stasis. His chief symptoms were continuous bilious vomiting, dilated pupils, and occasionally a curious nervous discharge, unlike, in general character, a true epilepsy. Grave doubts were entertained with regard to his recovery, acute tubercular meningitis, or tumour of base, being suspected. Under rigid treatment he recovered, and at the expiration of three months, vision, which during the attack had materially suffered, returned. I have now to relate the most interesting point of the case. On the 10th day of April of the present year this patient, now an adult, consulted me privately for confirmed epilepsy, giving the following details:—

After leaving the Hospital in 1873, he remained apparently quite well until the spring of '78, when, from no apparent cause, he began to decline in health, lose flesh, and become nervous. This was followed by cerebral neuralgia of an intensified form, which was in turn superseded by epilepsia gravior. The paroxysms are described as very severe, occurring at intervals five times daily. When under treatment for the earlier illness as a boy, the nystagmus was but slight; now it is most marked. The length of time which elapsed between the first and second illness, the unstable condition of grey matter lying dormant for so long a period, in readiness to again depart from its normal (healthy) functions from a cause difficult in this instance to define, renders the case one of more than ordinary interest.

In the form of minor epilepsy which characterised the early seizures of this boy, may we not infer that, though the cells were not in an actually diseased state, it was but a condition —a departure from healthy life—which portended ulterior disease? Then again, as in this example, how many cases of petit mal pass into the graver form without eliciting but a passing notice? I believe many more than we suspect.

It has never been my lot to diagnose the earliest changes in grey matter which ultimately lead to epilepsia gravior; but I suspect that were it possible to recognise such change—that is, so soon as the cells gave the slightest evidence of abnormal function—urgent and well-directed treatment might be of such service as to end in the permanent health of the unstable matter. This is more or less evidenced by the case I relate of continuous bilious vomiting, with nervous discharges, which, although apparently cured, remained latent for years, ultimately to break out afresh and merge into the graver form of epilepsy. Here, although the nervous discharges were frequent in the earlier illness, yet it is probable that the unstable condition of the grey matter was not advanced, and therefore the attacks were mild. Is it not possible, if the disease during these early stages remains unchecked, that the number of cells having a perverted function seriously increase until a large area of grey matter becomes involved?

Whilst jotting down notes for this paper, I was fortunate to meet with a case in which I truly believe the earliest symptoms of this form of disease were diagnosed. Three weeks ago I was asked by a medical friend to see with him a lady who was suffering from the following symptoms. Sudden sensations as of a weight being placed at the commencement of the spine immediately beneath the occiput. This feeling of weight lasted for a few seconds, and was succeeded by a violent throbbing, "trembling of the blood," she called it; then came flashes of light, rotary nystagmus, and a sudden falling forwards of the body, the fall being saved by seizing some article of furniture. Although entire unconsciousness never took place, yet so soon as the body fell forwards, there was an irresistible feeling to lie flat on the back. After remaining in the horizontal posture for ten or fifteen minutes the paroxysm passed away, to recur again in a few hours. The history was as follows. Very healthy and strong till a year ago, she then passed through a severe scarlet fever. Ever since convalescence from that disease these nervous discharges had shown themselves, and latterly with increasing severity. There had been on several occasions hæmorrhage

from the bowel; but as the system never suffered from it, and the general habit of the body was inclined to corpulency and the face somewhat flushed, I did not consider it a factor in the production of these nervous discharges, much to my medical friend's astonishment, who came armed to the consultation with that idea. He said he had carefully watched the case from the beginning, and it was a nervous system deprived of proper nourishment from that cause. I found the treatment had been chiefly iron and ammonia, which, taken in large doses, appeared to aggravate the symptoms most unpleasantly. Thirty grains of bromide of potassium, with 30-drop doses of tincture of digitalis, taken early before rising and late before retiring for the night, had a magical effect, the third dose subduing all the worst symptoms, and its continuance—thus far—keeping the patient in perfect health. This, I take it, was a case of the first degree. Here, I think, undoubtedly we had the earliest departure from healthy nervous action, the merest instability of nerve-cells which, left without appropriate treatment, would have passed on to grave functional disturbance and so on to epilepsy.

I have mentioned a case of disseminate sclerosis with nystagmus, and as disseminated sclerosis is, as a rule, followed at a late date by nystagmus, I must relate the history of a case now under my care.

Five or six years ago a gentleman of very active business habits suffered a serious loss which produced unusual mental grief, so much so that business was entirely neglected, and his family, as it were, deserted. His wife, who has great faculty for detail, assured me that for the whole of their previous married life he had shown unimpaired nervous energy, and was known to his friends and acquaintances as a man of more than ordinary stamina. Eighteen months after his business loss, his wife noticed that there was a slight dragging of the lower extremities and a decided uncertainty of gait. This symptom gained ground but slowly, and is not now very serious. The upper extremities were next affected. This paresis of the limbs remained for a long time as the only sign of disease going on in the spinal cord, and as it made but slow progress, his family were in hopes that he might recover.

This was not to be. Thirteen months after the first symptoms of paralysis, his wife noticed that he had a laboured manner of enunciation which slowly grew worse. She expressed it as "taking great pains to utter each word distinctly." Diplopia —transient at first, afterwards of a more permanent character —brought him under my care. Nystagmus was not then present, nor did it occur until some time afterwards. The nystagmus, transient and feeble at first, is now persistent and marked. In this case there is considerable interference with the retinal circulation, the discs being white with diminished calibre of arteries, and considerable dilatation of veins. Concerning the ophthalmoscopic appearances in nystagmus, I shall say more hereafter. The very gradual invasion of the nervous centres in this case renders it probable that a lengthened period will elapse ere a fatal termination arrives.

An interesting fact remains to be noticed concerning the apparent atrophy of the optic discs. To all apparent observation, it would seem as if from invasion of the optic tracts or chiasma, total destruction of nerves had resulted, and so total abolition of function. As a rule, this is not the case, arising from the fact that only a portion of the nerve suffers from a sclerosed patch. In other cases, the whole nerve apparently suffers, but not microscopically so, the medullary sheath having alone suffered, leaving the axis-cylinder intact.

To the fact that atrophy (apparent) of the optic disc is not correlated with total abolition of function in all cases, I have, in years gone by, drawn attention. *Vide* papers by me in the 'Lancet,' 'Ophthalmic H. Reports,' and 'Dublin Quarterly Journal of Medical Science.'

At that time pathological investigation had not thrown sufficient light on the subject for me to state, without fear of contradiction, why one apparently atrophied disc should be associated with complete visual loss; whilst another, differing in no wise, so far as the ophthalmoscopic appearances went, should suffer but slightly, and vision be sufficient for ordinary purposes.

That disseminated sclerosis in a large number of cases is followed by nystagmus is beyond doubt, as evidenced by

the writings of Charcot, Moxon, and others.[1] I have seen, I believe, but four such cases. These cases but rarely come within the range of ordinary ophthalmic practice; and as the nystagmus is a symptom which manifests itself during the late stages of the disease, we miss seeing a number of interesting cases which would afford valuable clinical instruction.

The inability to regulate visual co-ordination arises from many causes, but from none more so than direct injury to the medulla oblongata. Many years ago a gentleman, to all appearance enjoying robust health, whilst performing one of nature's functions, horrified the female who suffered his embraces by suddenly rolling on his side speechless. In less than half an hour from the seizure I was with him. The prominent symptoms were stertorous breathing and horizontal nystagmus, with widely-dilated pupils and distended palpebral orifices. In this case there had been sudden bleeding from a large vessel, and considerable destruction of tissue in the medulla. Unfortunately, the injured part was not obtained for further examination, so that the more exact lesion could not be determined.

A similar case to the above occurred some years ago, and from a similar cause. This brain was preserved, and is now, I believe, in our Medical School. The injury in this case was also destruction of tissue in the medulla from hæmorrhage.

Another case of a similar character, but if possible more interesting, occurred in the case of a lady now resident in this town. During, as she believed, perfect health, she suddenly fell down comatose whilst performing some household duty. When first seen by me her symptoms resembled in every particular those of the case first related, with the exception that the nystagmus was transient, occurring at intervals of five and ten minutes, and lasting for varying periods of from ten to forty seconds, the horizontal movements of the eyes being violent in manner from the way in which the muscles acted. In consultation with one of my hospital colleagues, it was decided to bleed freely. This was done to, say the extent of sixteen ounces, with a benefit very extraordinary; for while

[1] Consult an interesting paper by Dr. Moxon, 'Guy's Hospital Reports,' 3rd Series. vol. xx. 'Insular Sclerosis of the Brain and Spinal Cord.'

yet the blood flowed, the coma passed away, the nystagmus ceased, and immediate danger was averted. Although there was a slight relapse, the symptoms recurred in a minor degree, the patient recovered, and perfect convalescence resulted.

A case of injury to the medulla oblongata, followed by nystagmus, is ably reported in the 'Medico-Chirurgical Transactions,' vol. xlvi., 1863, by Dr. Waters. A young man received a severe blow on the left side of the face, followed by dilated pupils and nystagmus. The following day he walked to the hospital. Although he presented no very urgent symptoms, he suddenly expired the day following. A blood-clot occupied one portion of the medulla, and the right restiform body was lacerated. The grey matter of the floor of the fourth ventricle was also involved. This case illustrates how serious a lesion of the nervous centres may be without causing prominent objective symptoms.

I must mention a certain form of nervous disorder which I have had occasion to treat on six separate occasions, two of the cases showing marked diplopia and nystagmus.

The first case which I saw occurred ten years ago, and the patient, an engineer, is still under my care. The symptoms, although approaching in character those which we frequently meet with in minor epilepsy, are, when analysed, different in degree. Until lately I have been puzzled to name this disease, and have been content to term it "cerebral pulsation." The six cases I have seen occurred in men. The first was a civil engineer, the second a timber merchant, the third a man constantly engaged in literature, the fourth a general merchant, the fifth a clergyman, and the sixth a medical man in a large general practice. I mention the occupation of these men to show that the employment of each was one requiring brain work, and that of considerable strain in each case. I am not now referring to that form of passive cerebral congestion which is almost daily met with in over-worked business men, easily cured by rest and quiet and change of scene; but a condition, the nature of which in many particulars resembles the minor epilepsy, although totally different in others.

Notes of the first case seen furnish me with the following particulars:—Mr. C., aged 37, engaged as the principal

draughtsman in the office of a firm of civil engineers, in consequence of a large foreign order, had been drawing plans for many hours each day for several months. The success of the contract depended upon his skill, and as his father was one of the partners in the firm he represented, it made him doubly anxious to succeed. Naturally of a nervous temperament, the strain was too much, and one day, when passing from the office to the workroom, he was seized with sudden giddiness, and for a moment unconsciousness. Neglecting such a warning, he still pursued his usual avocations, until a paroxysm more serious demanded medical aid. On visiting the patient I found a small man with a well-knit frame, and what was quite unusual to his ordinary appearance as of six months previous, a face swollen and flushed.

He gave the following history :—" For months I have had a growing fulness about the brain, as if the blood-vessels were too small for their contents, and that the blood was endeavouring to find an outlet and the vessels were determined not to accommodate themselves to this new state of things." He had frequent hallucinations and impaired memory, the latter ailment being a great source of trouble, as he was frequently forgetting where he had deposited certain papers of importance, and otherwise doing unbusiness-like acts. It is hardly necessary to mention that with such cerebral plethora he had constant headache. Another peculiar symptom which alarmed him was that he suddenly fell forward when walking (this generally occurred soon after leaving an omnibus or railway carriage in which he had been journeying), but always managed to save the fall, by seizing hold of some adjacent object.

On many occasions these seizures were associated with unconsciousness, and always with horizontal nystagmus. The inability to regulate his visual co-ordination would often last for many seconds after consciousness returned, and was a perpetual source of annoyance to him, chiefly from the fact that many of those who had witnessed the paroxysm assured him that it must have been a real fit, " for your eyes rolled from side to side." Other than a disinclination for food, and a somewhat impaired digestion, I could not detect any disturbance of organs. The heart was apparently healthy, but

the arteries were hard and tortuous, having evidently undergone change which materially affected the circulation generally, as evidenced by the sudden fits of unconsciousness, due no doubt to cerebral hæmorrhage.

One peculiarity in the case was that he suffered from traumatic stricture of the urethra—not a bad one, but sufficient to annoy him, and require the passing of a metallic bougie monthly. He had an idea that the cerebral congestion was worse immediately before the time expired for the passing of the bougie. The further history of the case proved that this was not so. One curious phenomenon was a nervous discharge through the retinas of a series of bright flashes, as he described "just as if a number of matches had been struck one after the other within the eyes." Examined with the ophthalmoscope, the optic discs were markedly hyperæmic, the veins enormously distended and tortuous, and a condition of arteries seldom seen, they being hardly discernible from the veins, as if they were carrying venous blood, and also being of a calibre so large as to almost deceive you into the belief that they were actually veins much distended.

The habits of the patient were strictly sober in all things, and he had never contracted syphilis. As medicine and rest proved of little avail, I desired the advice of my friend Dr. Crichton-Browne, then of the West Riding Asylum, who agreed with me that the disease, although resembling the less serious form of epilepsy, was not really that disease. As the treatment adopted after consultation did not prove curative, I at last persuaded him to journey through the United States and Canada. The effect of the sea voyage was very remarkable, and he returned all but convalescent. By strict care and frequent rest the attacks did not recur more than once in six months. He has just passed through a rather severe one which confined him to the house and bed for four days. I have but little doubt the paroxysms would resume their old frequency and virulence should he again return to constant employment.

The other case in which nystagmus was present occurred in a general merchant whose habits were vicious, and whose constitution had undergone much wear and tear. This case

differed from the last in an important point. The hallucinations of the first patient were harmless. In this patient's case they assumed a dangerous form. Watching and restraint alone prevented him doing bodily mischief to his wife, who for the time the cerebral congestion remained was an object of extreme hatred.

Another cause other than excessive mental strain is a dyspeptic condition of a low type. The ill-nourished nervous matter from impurity of blood is probably an early factor in the production of the disease. I feel convinced from the lengthened character this disease generally assumes, and its not being very amenable to treatment, that it may become an entity—a disease which from its symptoms and duration will find a place amongst the ordinary nervous derangements of authors. An analogous form of the disease has been described as cerebral hyperæmia by Dr. Milner Fothergill in the West Riding Asylum Reports, by Dr. Hamilton of New York ('Nervous Diseases, their Description and Treatment,' 1878), and lately by Dr. William Hammond of New York. A condition of the retina is noticed by the last writer upon which I have always laid great stress in these and somewhat similar cases. There is a condition of passive congestion more or less always present, and a peculiar arterial fulness—such as I have just described—which is rarely observed in ordinary cases. Another abnormal condition of the retinal circulation is the extreme tortuosity of the vessels, an appearance which would lead one to infer a diseased condition of the arteries of the brain.

I must now pass on to describe a form of nystagmus with which I have long been familiar, and which I have described elsewhere. This form of the disease I have designated "Miners' nystagmus," from the fact that it occurs in men following the occupation of colliers. In giving a general description of this form of the disease, I cannot do better than extract from my former paper on the subject a few preliminary remarks:

On a Peculiar Form of Nystagmus.

"The history and symptoms differ but little in the cases already observed, but a fact worthy of special note is, that all the

patients as yet examined are by occupation colliers. The disease is ushered in by no premonitory symptoms, is persistent, not amenable to treatment, and usually occurs after the age of twenty-one. Further than the oscillation of the globes, which is usually a horizontal one, no trace of disease—either local or constitutional—can be found. The eyes are normal in shape and colour, the pupils act well, and the muscular movements are performed naturally, when the body is in the erect posture. One peculiar feature of the disease is the influence which the position of the body has over it. In the stooping posture—the position of a miner when at work—the oscillation of the globes occurs; as soon as the body is erect, the eyes cease their unnatural movements. Family history does not point to the inheritance of cerebral disease. Syphilis is not a factor in any one of the cases. The urine is healthy, the eyes present no ophthalmoscopic appearances of disease, and the power of vision is perfect. In some patients the globes oscillate as soon as evening advances, but this is an unfrequent symptom. I was at first impressed with the idea that pigmentation of the retina lay at the root of the mischief, but no single case under my care has presented any disease of that structure. The following is the history of one case. The patient describes the commencement of the very first attack he ever had thus: 'I was stooping at work, being quite well in health, and my eyesight good, when suddenly the light from my lamp appeared to be moving to and fro, and objects became indistinct. When I stood up, the sight returned. This peculiar condition has always since occurred, when I have stooped to begin work; and my eyes have rolled from side to side so long as I have continued with my body bent. As soon as I stood erect, the rapid movements of the eyes ceased, and clearness of sight returned.'

"This man I purposely admitted as an in-patient to watch his case, and try various kinds of treatment. Iron, strychnine, arsenic, quinine, and other drugs were taken without the slightest benefit. He was therefore made an out-patient at the end of six weeks."[1]

For several years after the publication of this paper I had

[1] 'British Medical Journal,' Jan. 3, 1874.

seen but few of these cases. I am indebted to the kindness of our present House Physician, Dr. Barrs, for having lately transferred to my care several cases of this disease.

During my early investigations into the nature and cause of this singular and interesting disease, I was much struck with the fact that each case appeared to be the exact counterpart of its fellow, that the disease occurred without apparent cause, and the pathological bearings were difficult of diagnosis. The last series of cases under my care have thrown considerable light upon the whole nature of the disease, and I therefore feel constrained to relate somewhat minutely its clinical history. In my earlier batch of cases the ophthalmoscope, to my intense astonishment and regret, aided me not at all. This I now ascribe to the fact that the cases then seen by me were of early date, the patients having applied for medical aid so soon as the disease appeared and before structural change (intra-cranial) had become sufficiently complete to affect the retinal circulation or affect in any way the nutrition of the optic tracts. Another and more potent reason of failure to detect pathological change was the short time the patients remained under observation; expecting, as they did, immediate relief and not receiving it, determined them to seek and if possible obtain it elsewhere, and so I was deprived of the opportunity of further clinical observation.

In three out of the number of miners now and lately under my care, the disease has been of some standing, and therefore I have been able to arrive at a more correct conclusion as to its etiology and pathology.

I will relate the history of one case now under my care, which is more or less typical of the rest.

March 11, 1880.—W. W., æt. 44, states that three years ago his visual co-ordination was affected in the following manner. He was stooping with his head bent towards the right side, getting coal, when suddenly the light of his lamp appeared to be moving rapidly to and fro. Much alarmed, he at once assumed the erect posture, when, on again looking at the lamp, the light was stationary. The account of the commencement of the disease is the same in each case I have yet met with. The nystagmus is sudden, never preceded by

symptoms of ill-health, and afterwards is persistent when the miner is at work, but in many cases, indeed in most, the patient has power when in the erect posture to regulate and control the muscular action of the eyeballs. On questioning, I elicited further facts, which were that the day following the first attack of nystagmus, as he was preparing for work, he was attacked by a curious sensation which he said arose at the pit of his stomach and passed upward through his chest and neck towards his head (epileptic aura). He then lost consciousness for a few seconds, and when reason returned, he was surprised to find he had unlaced one of his boots which he had immediately before fastened preparatory to leaving home for the pit. Since this attack the fits have recurred at intervals with nearly always the same result, viz. the removal of some article of dress, either his coat, waistcoat, or necktie. The nystagmus is constant when at work, but in this case the movements of the globes (horizontal) are performed but a few times in each minute, and consequently he can follow his occupation with moderate success. This is the only case I have met with where the patient can continue his employment after once the nystagmus has become persistent. In this case, as in one other, if the patient is cognisant of the approaching fit he can, by quickly swallowing his saliva, prevent it, and he can also ward it off by taking a deep inspiration. In the history of this case, as in that of each one I have hitherto gone into, there is not a single factor which we so frequently meet with in cases of a somewhat similar kind, as a cause, such as syphilis, tumour of brain, meningitis, tubercle, cerebral lesion from injury, typhoid fever, &c. The history in one and all is remarkable for the absence of inherited troubles, and the sufferers have as a rule been strong and vigorous prior to the attack.

I have during the past six months seen six well-marked cases of miners' nystagmus of long standing, and in each patient the instability of the muscular apparatus is associated with a low form of epilepsy (petit mal). In the less-marked case the nervous discharges are so slight that without careful study they might be overlooked.

These seizures, however slight, do undoubtedly form one

group of minor epilepsy which is very frequently clinically neglected and very frequently misunderstood, the treatment being directed to the digestive tract solely; the supposition being that the brain is simply at fault, because it is supplied with blood of a kind insufficient for its proper (healthy) nutrition. This is not so; the two diseases are totally dissimilar. The brain of the confirmed dyspeptic is never benefited by the exhibition of a drug likely to increase the stability of the nervous centres, such as bromide of potassium or Indian hemp; but, on the other hand, is much benefited by drugs which put the general digestive tract into good working order.

The real cause of the disease is difficult of explanation. The men aver it is due to the fact that they are frequently working up to their waists in water for long periods, and that the difference in the workings has a material effect upon their constitutions.

Some pits are notorious for the amount of water they contain, and also for the number of men who are habitually ailing. Other pits are characterised as the healthy ones, from the fact that they are practically free from damp, and the men working in them are but seldom ill. I have but little doubt that this statement is truthful, and as wet and cold are the immediate cause of so many serious diseases of the spinal cord, we may, I think, say that the frequent immersion of the lower half of the body in water is the primary cause of the disease.

One fact we must not lose sight of, which is that in miners' nystagmus the oscillation of the eyeballs only occurs when the miner is at work and when the head and neck are much bent. This fact leads us to the belief that the particular area (medulla) is, so long as the neck is thus strongly bent, in a condition of venous engorgement, consequent upon the pressure on the large blood-vessels of the neck; and as the pressure is constant—that is, constant so long as the man remains at work—there is a continuous cell-discharge from unstable grey matter, rendering visual co-ordination impossible. The fact that so soon as the patient assumes the erect posture the nystagmus ceases, renders it clear that venous engorgement is the chief factor in this curious phenomena.

I copy from my note-book the ophthalmoscopic appearances of the fundus in each of the last eight cases of miners' nystagmus:—

CASE 1. The discs are red. Marked venous stasis.

CASE 2. The disc of the right eye is normal, but the veins carry too much blood; the disc of the left eye is pale, and the venous stasis very marked.

CASE 3. Hyperæmia of discs. Vessels rather too full.

CASE 4. Discs natural. Veins very tortuous and distended.

CASE 5. Discs anæmic. Vessels normal.

CASE 6. The appearances are normal in each eye.

CASE 7. Hyperæmia of discs; veins singularly tortuous and distended.

CASE 8. Discs red. Veins distended. Arteries, in comparison, very small in outline; possibly normal in calibre, but they appear small in close proximity to distended veins.

With but few exceptions, the ophthalmoscope disclosed a venous stasis which is always more or less persistent. In a few cases opposite conditions of the disc are noticed: in some cases we find anæmia, in others hyperæmia, and occasionally the disc appears normal.

Treatment proves but of little avail. I have tried many drugs, and have continued their use for long periods, but have never as yet met with success. The nearest approach to success was in the case of a man who suffered from a very exceptional form of miners' nystagmus (the oscillation being continuous). The liq. strychniæ had a speedy and marked effect in checking the undue movement of the globes whilst he was away from work. On resuming work, as soon as he bent his head and neck the nystagmus returned, and he was compelled to again leave his employment.

ON MUSCULAR SPASMS KNOWN AS "TENDON-REFLEX."

BY AUGUSTUS WALLER, M.B.

MUCH attention has of late been conferred on a certain class of spasm symptoms, viz. tendon-reflex phenomena and cloni. Their clinical import is an independent fact, their physiological interpretation has hardly yet come to rest in a satisfactory generalisation. First demonstrated for the extensor cruris by percussion of the ligamentum patellæ, the demonstration has been extended to most of the prominently acting muscles of the body.

To commence with the best-known instances, let us consider the "knee phenomenon" and the "ankle-clonus." These are spasms elicited—the former by percussion of the ligamentum patellæ—the latter by sudden passive flexion of the foot. The former is seen as a single, the latter as a multiple spasm, and they are commonly found associated in threatened, incipient, or progressing contracture. The first question, therefore, that suggests itself is: wherefore a *single* rectus-spasm and a *multiple* gastrocnemius-spasm?

Assuming that the effect on the muscle is brought about by a pull of its ligament,[1] the answer is obvious. At the knee the single blow is the single stimulus to a muscle which

[1] Tschiriew ('Arch. f. Psych. viii. 3) has shown that, with an uninjured tendon, percussion is the only stimulus that can excite the spasm; that this stimulus remains effective after ligature and crushing of the tendon, and after division of all nerves to the joint; that subsequently a sharp pull of the cut tendon still excites the spasm. Dr. Gowers' observations point to the same conclusion. Further, electrical stimulation of the ligamentum patellæ does not provoke any muscular reaction; and in investigating cases where there is no reaction to tendon percussion, the difference between a blow on the patella or on its ligament can be felt by the hand placed on the rectus. In the latter case, vibration of percussion is far more distinctly perceived.

contracts and then remains quiescent; at the ankle the first contraction excited by the tension of passive flexion restores the mechanical readiness to a second tension stimulus by passive flexion—the reactions of the muscle break a continued flexion into successive jerks, each jerk causing a contraction, each contraction making ready for a jerk. A clonus or series of spasms thus obtains which is not to be classed as a rhythm by continuous influence.

Just as the exaggerated phenomena are commonly associated in disease, so are their normals commonly associated in health. The kick provoked by a blow just above or below the knee and the dancing of the leg are tricks of boyhood, and every one knows how suddenly a man can be pulled down by a blow across his hamstrings. We see in them the same difference of "façon d'agir" as in their clinical congeners—in the one case a single tensile jerk to the tendons of thigh-muscles, in the other a tensile jerk to the tendon of the calf-muscles, repeated at each fall of the limb.

The identity of the mechanism is further established, inasmuch as: 1st. A single tap on the tendo Achillis of the *free* foot causes a single extension of the foot. 2nd. Percussion to the rectus of a *fixed* leg, evokes both in normal and abnormal cases, not always a single spasm only, but sometimes a short series of two, three or more spasms.[1] 3rd. In cases of abnormal exaggeration, a knee-clonus may sometimes be demonstrated, of the same spasm-frequency as the ankle-clonus. In some normal cases the clonic tremor with which we are familiar in muscles crossing the ankle-joint, may be imperfectly imitated in muscles crossing the knee-joint, by placing the leg so that the relation between tendon-extension and weight to be lifted will allow the falling weight to excite the contraction that raises it again.

Muscular contraction, provoked by muscular extension, is

[1] This may be explained as follows: The elastic extensibility of muscle is greatest during contraction. If it contract while both its insertions remain fixed, its actual length is unaltered, while physiologically it is extended by the resistance to its own contraction. Thus it is, that percussion of the ligament of the fixed rectus causes (1) contraction in response to tensile jerk; (2) contraction in response to the momentary passive hyper-extension at cessation of contraction.

the element common to all these phenomena; any blow on tendon, or muscle, or near muscle, that excites the muscular fibre, does so by *extensile vibration.*

Knee-clonus, as a clinical symptom, is frequently to be found in cases where the ankle-clonus is pronounced. It may be produced, sometimes by a flexing jerk of the extended limb, sometimes by percussion of the ligament, or muscle, or patella, and it sometimes supervenes on an attempt to extend the leg, or in continuation of a single excited spasm, when its spasm-frequency is, of course, diminished by the weight of the oscillating limb. It is necessary that there should be a certain balance between muscular contraction and resistance to contraction, in order that the movement initiated by any tensile jerk shall be repeated by sudden restoration of extending resistance. The foot lends itself to the setting of this balance far more aptly than does the leg, and it is more difficult, on a patient, to demonstrate knee-clonus than ankle-clonus, or on oneself, with unenhanced irritability, to set going a knee-clonus than to arrange the foot to dance. Perhaps the easiest method to get a knee-clonus is to tap the patellar tendon of the leg fixed at a certain angle of flexion; a short series of spasms will appear in the rectus, presumably when the conditions allow of reverberation between contraction, and momentary extension on cessation of contraction. By extending the leg for an instant, and allowing it to drop slowly, it may be noticed that as it passes through a certain angle of flexion, breaks in the uniform movement occur; just as in the slight nystagmus of debility, lateral jerks are seen at a certain ocular angle, which disappear with further effort, and reappear on its gradual relaxation. Such tremor, whether it be in leg, or eye, or other part, is a rudimentary instance of the more striking and easily excited clonus, or nystagmus, or epileptiform spasm of morbid irritability. It is possible for a brief period voluntarily to maintain the leg at that angle where extension by weight and weight-sustaining contraction shall be so related as to reverberate. But the conditions to a continuance of such motion are not favourable; the limb tends to fall out of the correspondence between muscular extension and muscular contraction, which is the condition of the rhythm, i.e. there is too

much weight, too little contraction; or it is raised out of the correspondence by an excess of voluntary contraction, i.e. there is too little weight, too much contraction. When, however, the muscle has become fatigued by more prolonged extension, this excess of contraction over weight becomes removed, and the clonic tremor may be seen to supervene in the completely extended leg.

The following table contains the condensed notes of cases hearing on points in question, with time measurements of some of the phenomena.

No.	Case.	Phenomenon.	Time.
I.	*Right Hemiplegia* . . .	Patellar tap to rectus-contraction	R. ·04″ L. ·045
II.	*Cerebral Disease.*—Double optic neuritis. Tumor?	Patellar-tap to rectus-contraction . . . . . Front-tap to gastrocnemius-contraction	·04 ·045
III.	*Syphilitic Paraplegia* . .	Patellar-tap to rectus-contraction	·035–·04
IV.	*Paraplegia*	Ditto	R. ·05 L. ·05
V.	*Anterior Poliomyelitis.*—Exposure during Zulu war; no contraction in response to percussion at any part other than left wrist; reactions of degeneration.	Wrist-tap to wrist-extension	·0525
VI.	*Normal*	Patellar-tap to rectus contraction	·05
VII.	*Right Hemiplegia* . . .	Ditto	R. ·05 L. ·0475
VIII.	*Double Sciatica*	Ditto	R. ·05 L. ·05
IX.	*Locomotor Ataxy* . . .	Ditto	R. ·055 L. ·055
X.	(?)	Ditto . . . . Front-tap to gastrocnemius-contraction	·0425 ·04
XI.	*Spinal Syphiloma.*—On left side, no reaction to patellar-tap, no knee-clonus, ankle-clonus, reactions to front-tap and Achillis-tap excessive. On right side, reaction to patellar-tap excessive, knee-clonus, slight ankle-clonus.	Patellar-tap to rectus contraction . . . . . . Front-tap to gastrocnemius contraction . . . . Achillis-tap to gastrocnemius-contraction . . . Knee-clonus, R. per sec. . Ankle-clonus, L. per sec. .	R. ·05 L. ·05 L. ·05 10 10

No.	Case.	Phenomenon.	Time.
XII.	*Disseminated Sclerosis.*—Right hemiplegia 13 years ago. On right side exaggerated patellar reaction, ankle clonus, knee-clonus.		
XIII.	*Left Hemiplegia.*—Right rectus reaction excessive. This was the only exception to the rule, viz. increased excitability on the diseased side. There may have been an error of measurement, or in my notes; or the reaction may have been becoming abolished. The case was lost sight of.	Patellar-tap to rectus-contraction	R. ″·45 L. ·6
XIV.	*Normal*	Patellar-tap to rectus-contraction . . . . . Achillis-tap to gastrocnemius contraction . . . Galvanic stimulus to rectus Galvanic stimulus to gastrocnemius	·036 ·04 ·033 ·033
XV.	*Progressive Muscular Atrophy*	Wrist-tap to biceps contraction	·05
XVI.	*Paraplegia.*—So-called spinal epilepsy. Any sort of impression is liable to induce violent spasmodic agitation. If patient extend his leg, it instantly enters into violent clonus, followed by clonus of the opposite leg, of the abdominal muscles, and of the upper extremities. The violence of the movement is such as to shake the bed and even the room. Cold or anger are particularly liable to excite the agitation. By firmly grasping the rectus, the clonus of that muscle may be arrested. The patient's friends have been accustomed to sit upon him, in order to arrest the general convulsion.	Ankle-clonus, per sec. . . Knee-clonus, per sec. . .	8 8

No.	Case.	Phenomenon.	Time.
XVII.	*Left Hemiplegia.*—It is noteworthy that a tap on radius provokes flexion of the forearm; a tap on ulna, extension of the forearm; a tap on knuckles, flexion of the fingers	Wrist-tap to biceps-contraction . . . . . Patellar-tap to rectus-contraction	" ·045 ·05
XVIII.	*Syphilitic Paraplegia.*—No response of rectus to patellar-tap on either side; on right side, foot is everted in response to a tap on the ligament, or on the patella, or on the tibia; at the same time the hamstrings are felt to tighten.		
XIX.	*Right Hemiplegia and Aphasia.*—Late contracture in active state. A tap on any bony point of right arm or on clavicle provokes violent flexion and extension, chiefly of the arm, frequently passing into a rapid true clonus of the whole extremity, lasting about 30 seconds.		
XX.	*Left Syphilitic Hemiplegia.*—All reactions are increased on left side. Excessive reactions of several muscles of upper extremity to tap on wrist. It is noticeable, however, that with tap on radius, or radial side, a flexion of the arm is evoked; with a tap on the ulna, or ulnar side, an extension; tap on knuckles excites flexion of fingers.		
XXI.	*Traumatic Paraplegia* . .	Ankle-clonus, per sec. . . Knee-clonus, per sec. . .	8 to 9 10
XXII.	*Traumatic Paraplegia* (child).	Ankle-clonus, per sec. . . Knee-clonus, per sec. . .	10 9

It obtained throughout the above cases that a greater irritability of muscle to extensile vibration is associated with lesions that isolate spinal segments from superior influences; despite of the usual association with contracture it does not necessarily follow that each reaction is from the emancipated centre, but only that some influence from the centre (viz. the reflex tonicity maintained by the spinal cord) normally furnishes to peripheral elements, muscular or intra-muscular, a *condition* of the reaction. Abnormally under excess or defect of such influence the muscular reaction may be enhanced or fail.

To resume the identification of the phenomena at the knee and ankle-joints, let us consider the round numbers of their time estimates. They are

Achillis-tap to gastrocnemius spasm . . .	·03″ to ·04″
Patellar-tap to rectus spasm	·03″ to ·04″
Ankle-clonus	8 to 10 per sec.
Knee-clonus	8 to 10 per sec.

These numbers plead in favour of the argument that the mechanism of analogous spasms at the two joints is identical. We have seen cause to attribute throughout the same stimulus to both kinds as types of a class, therefore the mechanism of all the above spasms is probably identical.

With regard to the question common to them all, namely: Is the mechanism wholly peripheral or reflected from spinal centres? (1.) Tschiriew finds for the quadriceps of rabbits, that the reaction is abolished after section of anterior roots, or of posterior roots, or of that limited portion of the cord whence the crural nerve proceeds, even if at the time of tendon-percussion, muscular tension be maintained by moderate faradisation of the peripheral nerve stump. (2.) The clinical records of neurologists (Westphal, Erb, Buzzard, Grainger Stewart, Charcot, Gowers, Bramwell, &c.) prove that the abolition of the reaction corresponds with incapacitations of spinal matter (e.g. ataxy, infantile paralysis), that its exaltation is associated with interruptions above grey matter, and is especially a token of irritability, in a centre undergoing the irritation of degeneration (e.g. paraplegia, lateral sclerosis). (3.) As the effect of strychnia, by which the irritability of spinal centres

is enhanced, muscular reactions increase in vigour. (4.) The latent interval between stimulus and contraction is presumably greater than that of a muscular latency. (5.) Contractions identical with those by percussion of tendons, may be provoked by percussion of remote parts, e.g. bony joints, and in some cases crossed contractions are elicited.

By data (1) (2) and (3), no more is proved than that a certain influence from spinal centres is necessary to the reaction of muscles by the percussion of their tendons; (4) is of no value—if a period of ·03″ (*vide* note, page 187) is above the presumable value of direct muscular latency, it is also (as Dr. Gowers has remarked) below that of a reflex event. Helmholtz's original experiments gave for the nerve transmission-rapidity 30 to 90 m. per sec. according to temperature. Bloch has stated that the rate might reach over 150 m. per sec. My own measurements assign to it under ordinary conditions a value between 30 and 50 m. (*vide*

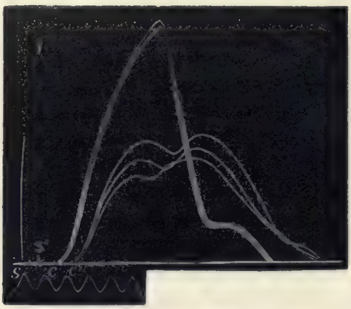

FIG. I.—NERVE TRANSMISSION-RAPIDITY.

Fig. I.). Therefore, roughly speaking, to take rectus and gastrocnemius respectively ½ and 1 m. distant from their

Fig. I.—Electric latency of a thumb-index pinch, recorded on the revolving cylinder by a lever-bearing tympanum, with which was connected an explorer between thumb and index as "pince myographique." Quick rate of cylinder – 3 mm. = $\frac{1}{100}$ sec. – tuning-fork tracing 100 vv. per sec.

$s - s'$. Physical latency of media.
$s' - c$. Latency of stimulus to median at bend of elbow.
$s' - c'$. Latency of stimulus to brachial nerves above clavicle.
$c - c'$. Difference, expressing time occupied by nerve-impulse between these two points.

N.B.—S to s' is the physical latency of the transmitting media ascertained by

spinal centres, the nerve-delays should be about ·02″ to ·03″ and ·04″ to ·06″, to which have yet to be added the central and muscular delays which make up the total reflex in-

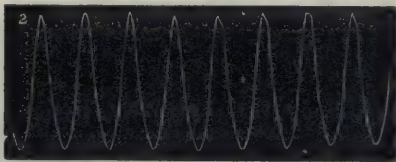

FIG. II.—KNEE-CLONUS.

terval. The gastrocnemius reflex latency ought to be sensibly greater (about ·02″ to ·03″) than the rectus latency,

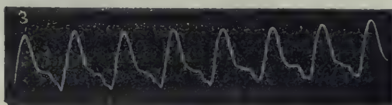

FIG. III.—ANKLE-CLONUS.

whereas I have found it to be about the same (·03″ to ·04″. *Vide* Figs. IV. and V.). And finally I find that the direct latency of the gastrocnemius and rectus to galvanic

independent measurement. Stimulation made by contact of metal-point with knife-edge on revolving cylinder, whereby current is closed and opened. Instant of contact s recorded by a contraction while cylinder is stationary. Intervals between instant of contact and contraction $s - c$, $s - c'$, shown with cylinder at full speed. Taking the intervening length of nerve at 30 to 35 cm., the interval (in this tracing slightly less than $\frac{1}{100}$″) denotes a nerve-transmission rapidity between 40 and 50 metres per sec. In the tracing figured, three successive contractions of each stimulus were allowed to coincide, as a testimony of mechanical accuracy. Tracings, from which estimates were actually taken, were made fine enough to allow of microscopic measurement.

Fig. II.—Knee-clonus (No. xviii.); Fig. III., ankle-clonus (No. xviii.). (Medium rate of cylinder, 5 cm. = 1 sec.)

NOTE.—Tschiriew estimated the percussion-contraction interval at ·033″. Dr. Gowers estimated it at ·10″; for the "front-tap" he found it to be ·04″ to·05″; for the ankle-clonus he found a spasm frequency of 6 to 8 per sec., for a knee-clonus of 2·5 per sec. From this he is led to the belief that at the knee the phenomenon is reflex (there being also a slight indication of a direct contraction), while at the ankle it is direct. Measurements that I made to control the discrepancy led me to assign ·04″ as the mean latency of the knee-phenomenon, while for the knee-clonus I found a spasm-frequency the same as for the ankle-clonus (viz. 8 to 10 per sec.) (*vide* Figs. II. and III.). I therefore concluded that Tschiriew's estimate is the more accurate one. There may have been a trifling excess in some of my first measurements (viz. ·05″), where percussion on a tube across the ligament made the signal, due to a minute delay suffered by the percussion vibration along

stimuli applied to their motor point is longer than has been assumed, viz. about ·02″ (*vide* Fig. VI.). (5.) When percussion provokes contraction it is by transmission of vibrations to

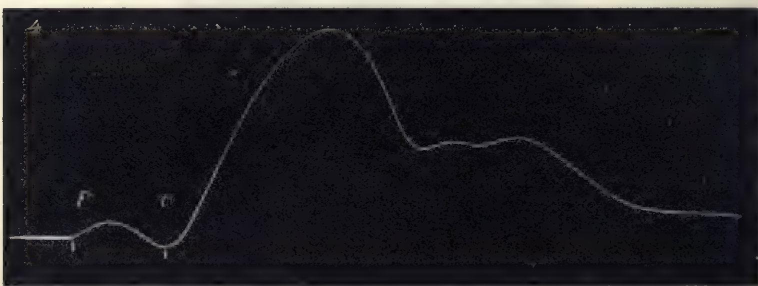

FIG. IV.—TENDON-PERCUSSION LATENCY OF RECTUS.

irritable muscles, their irritability may be so diminished that vibration viâ tendon is insufficient, and percussion directly to their substance is necessary to excite contraction (e.g.

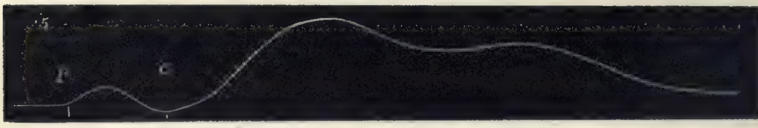

FIG. V.—TENDON-PERCUSSION LATENCY OF GASTROCNEMIUS.

ataxy). On the other hand their irritability may be so increased that the vibration by percussion of the bone into which

tendon to muscle—a medium of imperfect rigidity—or to a trifling advantage in time for the manifestation by the lever of a sharp percussion impulse over that of the more massive contraction impulse. M. Brissaud's recent measurements, with which I was not acquainted at the time, are nearly identical with mine, viz. ·035″ to ·05″.

Fig. IV.—Percussion latency of rectus (normal subject). (In this tracing 12 mm. of the abscissa denote the latency, viz. ·04″.)

P denotes percussion of lig. pat.

c denotes contraction of quadriceps.

Fig. V.—Percussion latency of gastrocnemius (normal subject).

P denotes percussion of tendo Achillis.

c denotes contraction of calf.

Recorded by a lever-bearing tympanum, in connection with an explorer fixed on the muscle. A tube, branching off the tympanum tube and crossing the tendon, was first employed to record percussion. But the jar communi-

they are inserted, or of still more remote parts, is competent to excite their contraction. This was well exemplified in Cases XVII. and XX., where by percussion of radius the fore-arm received a flexing spasm, while that of the ulna provoked an extending spasm. Naturally, along rigid bones vibrations most readily travel. I have frequently observed the same

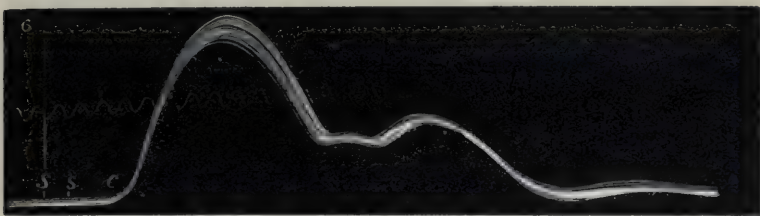

FIG. VI.—ELECTRIC LATENCY OF RECTUS.

to occur in weakly subjects (e.g. phthisis). With regard to "crossed reflex" though I have often seen clonus invade the opposite extremity, I have not witnessed any case where a simple spasm of rectus was undoubtedly crossed; at the most there has been adduction of the limb opposite to the one percussed. In such cases the excessive movement affects the trunk, and gives rise to an appearance of contraction in the opposite limb. Extent of agitation appears to be as naturally referred to extent of vibration in irritable muscles and vigorous contraction, as to spinal diffusion. (6.) The percussion-contraction interval is the same, viz. about ·035″, whether the substance or the tendon of the muscle be struck.

cated to the explorer by the percussion is sufficient (as in the above two tracings) to record its instant. It is also more satisfactory, since it eliminates certain sources of fallacy, e. g. time of vibration-transmission along tendon and muscle.

Fig. VI.—Electric latency of rectus (normal subject;) in this tracing the latency is ·02″ (quick rate – 3 mm. = $\frac{1}{100}$ sec.).

Stimulation made and marked as in Fig. I. Interval between instants of contact and contraction $s - c$ shown with cylinder at full speed. Fifty successive contractions coincide in this tracing.

$s - s' =$ physical latency of media.
$s' - c =$ neuro-muscular latency.

The ordinary provocatives of neuro-muscular contraction may be ranked as follows, in order of efficacy:

1. The galvanic or continuous current.
2. The faradic, or interrupted current.

3. Direct percussion of muscle.
4. Percussion of the tendon of a muscle.[1]

5. Percussion of the bone into which its tendon is inserted.
6. Percussion of still more remote parts.

If a muscle react to any one of these stimuli it will react to all preceding stimuli on the above list. If a muscle do not react to any one of these stimuli, it will also not react to all succeeding stimuli. The stimulus to which a normal muscle reacts ranges between 3 and 4. Its irritability may be increased so that the muscle reacts to 5, or to 5 and 6; or it may be diminished so that it only reacts to 2 and 1, or to 1, or not at all.[2]

Exceptionally it happens that at certain stages of altering irritability, non-response to one stimulus is associated with excessive response to a preceding stimulus—e. g. in Bell's palsy, as is well known, there is a period when reaction is 0 to faradaism, + to voltaic interruption; in ataxy Dr. Buzzard remarks that while reaction to tendon-percussion is 0, reaction

[1] According to the usual view, direct percussion of muscle provokes direct contraction—percussion of its tendon, reflex contraction. I find the percussion-contraction interval of the former to be ·03″ to ·035″; (i.e. the same as in the so-called reflex), and cannot therefore subscribe to the distinction. At the same time I fully recognise the value of the sign, the importance of which in the diagnosis of tabes Dr. Buzzard has made clear—viz. absence of "tendon-reflex," with muscular response to a direct blow, or to faradaism.

[2] Practical bearings of the above propositions are not far to seek. For instance, if a tap on the ligament or on the substance of any muscle cause it to contract, we know, *ipso facto*, that that muscle will respond to faradaism, and *à fortiori*, to galvanism; so that a fillip to the muscle will frequently spare the application of a superfluous electrical test. If it happen to a litigation patient that one physician should swear to the presence of tendon-reflex, or of mechanical irritability—say in the rectus muscle; while another should vouch for the absence of electric contractility in the same muscle, we know that the two statements are incompatible. The question is reduced to one of probability between affirmation of positive observation, and affirmation of negative. It is less likely that an observer testifying to a contraction seen and felt should be the victim of delusion, than that a witness to the absence of electric contractility should have failed to test or observe completely.

to direct percussion frequently appears to be +. Of these facts I do not attempt any explanation at present.

I am led to doubt the now generally accepted theory that the reactions known as clonus and tendon-reflex are subserved by spinal nervous arcs; while I admit as their highly probable "sine quâ non" the reflex tonicity exerted by the spinal cord. The fact remains constant that spinal conditions, by some influence, be it mediate or immediate, modify the response of muscle to extensile vibration. The foregoing paper contains facts militating against the theory in possession; it does not furnish data of theoretic construction, I have therefore no right to evolve gratuitous supposition. The question has interests broader than those involved in the interpretation of a clinical symptom; its discussion is reserved for a future occasion.

ON RIGHT OR LEFT-SIDED SPASM AT THE ONSET OF EPILEPTIC PAROXYSMS, AND ON CRUDE SENSATION WARNINGS, AND ELABORATE MENTAL STATES.

BY J. HUGHLINGS-JACKSON, M.D., F.R.C.P., F.R.S.,

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In this paper I speak of the epilepsy of nosologists, sometimes called "genuine" epilepsy, or epilepsy proper. In this epilepsy consciousness is either lost first of all or very early in the paroxysm. Loss of consciousness, as the first or nearly the first thing in a paroxysm, is, I presume, a sign that the central discharge begins in some part of the highest cerebral centres. In epileptiform seizures consciousness is lost late, and is not affected at all when the convulsion is very limited in range; in these seizures the discharge begins in some part of the lower cerebral centres (in some part of Hitzig and Ferrier's region). I think it is important to note, not only in epileptiform seizures but in epileptic seizures, on which side the first spasm or other abnormal condition is. Hence we ought to inquire whether the patient be right or left-handed. In many cases of epilepsy proper the spasm is for the most part bilateral, but rarely is it quite equal and contemporaneous on the two sides at its onset. In many cases the head or eyes or both turn to one side at the onset. We ought to try to get to know to which side the first turning is. Unfortunately the accounts given by the patient or his friends are often little trustworthy. Besides, sometimes the head first turns to one side and then to the other; I mean that sometimes turning to each side occurs before spasm spreads to the limbs or trunk, or, at any rate, before it involves either of these

regions in any marked degree. I had recently under care a case of convulsion in which, in one seizure, the head turned to one side at the onset, and in the very next seizure to the other. If we find, however, that the first spasm is always on the same side in every seizure, we may reasonably infer that the "discharging lesion" is of some part of the opposite cerebral hemisphere. In some cases there is no turning to one side; the eyes may go up and the head back. I suppose this is owing to great rapidity of the discharge.

I think the time has come for recognising that many different epilepsies are grouped under the one term epilepsy; we have come to recognise that at least several different epileptiform seizures are grouped under the term epileptiform. The paroxysm of epilepsy which begins by vertigo is a different epilepsy from that beginning by a sensation referred to or near to the epigastrium (solar plexus ?). The seats of the "discharging lesion" must differ when there are these two different paroxysms. There are, at any rate, as many different epilepsies as there are different warnings; the so-called warning is simply the first result from, or during, the central discharge; it is the thing of most localising value. Besides, it would appear to be very evident that the "discharging lesion" may be in either the right or in the left cerebral hemisphere. I wish to urge, Firstly; that we should study—not the epilepsy of nosologists, but each different epilepsy as distinguished by its particular warning. I fully believe that it is a good thing to take epilepsy to be a sort of clinical entity, and, for an example, to work up the "warnings of epilepsy." In this way we can say that of sensation "warnings of epilepsy," coloured vision is common, noise in the ear rare, and taste very rare. Such are empirical generalisations of value. Like other physicians I have long worked in this way. But to make rational generalisations we should also work in another way. Instead of speaking only of different warnings "of epilepsy," I think we should say, too, that there are different epilepsies each with its own warning, and some with no warning. Secondly; we should observe each epilepsy throughout, from the warning, if there be one, through the paroxysm, to the after condition of actions, when there are any. To begin with, we

must study groups of cases according to the warnings. This kind of study is extremely difficult. It is to be understood that some of the statements I put forward as results of such study are made with great diffidence as to their exactness. He who undertakes a task of this kind is sure to make mistakes, however careful he may be. This short paper, I may say, represents great labour.

I dare not state it as a fact, but my impression is that when an attack of epilepsy starts by vertigo, *in the sense of there being apparent movement of objects to one side,* the first part of the convulsion is usually right-sided, and that external objects appear to be displaced to the right. Such vertigo would signify discharge of some part of the left cerebral hemisphere. I have put this doubtfully, partly on account of the difficulty there is in getting trustworthy information from the patients and their friends, and partly because some observations made at my request do not support impressions from what I have personally gathered from patients. It may here be remarked that the term "vertigo" is often used somewhat loosely. I do not take the expression "giddiness" from a patient's mouth to always mean true giddiness. We have to put down not his name for, but the description he gives of, the sensation he calls giddiness. Unless we take pains to be accurate in our examinations as to the question propounded, our observations will be of little value. The investigator who simply asks leading questions on this and some other matters to be mentioned shortly is not accumulating "facts," but is "organising confusion." He will make errors enough without adopting a clumsy plan of investigating which renders blundering certain. I would expressly remark that by the expression "epileptic vertigo," a slight fit of epilepsy is not meant, but vertigo in a slight fit of epilepsy, or at the onset of a severe one—that the patient has the sensation of external things moving or of himself turning. I submit that it is inconvenient to use the term "epileptic vertigo" for slight epileptic paroxysms in which there is no vertigo.

Some patients have a "warning" of smell; others, a sensation referred to, or to the neighbourhood of, the epigastrium; others have, sometimes in addition to one of the above-mentioned crude

sensations, movements of mastication, or movements like those of tasting. With these warnings the first spasm, if there be any, is usually on the left side. I suppose the movements mentioned indicate discharge of centres for taste, although the patient has no sensation of taste. It is very rare to find any sensation of taste as a "warning" of any kind of seizure.

I must stay to remark on the movements above-mentioned. It is submitted that they cannot result directly from an epileptic discharge. An epileptic (that is a sudden and an excessive) discharge of the nervous arrangements for these movements would produce that contention of movements which is spasm, not movements properly so-called. I suppose they are indirect (reflex) consequences of discharge of nervous arrangements serving during taste. Mutatis mutandis for rubbing the hands (discharge of tactual centres?), and writhing of arms during suspended respiration, in other seizures. The hypothesis put forward is quite in accord with one of Ferrier's deductions from an experiment, is indeed a re-statement of it with application to disease. Ferrier ('Functions of the Brain,' p. 189) writes: "As regards taste, I think, that the phenomena occasionally observed in monkeys on irritation of the lower part of the middle temporo-sphenoidal convolution, viz. movements of the lips, tongue, and cheek-pouches, may be taken as reflex movements consequent on the excitation of gustatory sensations." In some slight cases of epilepsy the patient spits. I suppose no one would imagine such a highly compound movement to result (directly) from an epileptic discharge. It might, I submit, be plausibly put down as an indirect consequence of discharge of gustatory central nervous arrangements. So then in some slight fits of epilepsy we have not only that "clotted mass" of movements we call spasm, but also movements properly so-called. (I think too that there is in some cases, besides spasm, inhibition of lower motor centres and consequent paralysis.)

There is another way in which the warnings mentioned may be considered, as they are excessive and crude developments of objective or of subjective sensations. Unfortunately the terms "objective" and "subjective," so frequently used by medical men, are used in different senses. Thus, sometimes the term

"subjective" is used for psychical states in contrast to the accompanying nervous states, which latter are then called objective. Sometimes the term "subjective," or the subject, is used for mind, or even without distinction between mind and organism, in contrast to the environment, which latter is then considered objective, or the object. Sometimes the term "subjective" is used for a sensation internally initiated, and the term "objective" for a sensation peripherally initiated; thus, a patient who can smell nothing, and yet has stenches in his nose (at the onset of epileptic seizures, for example), is said to have no objective sense of smell, but to have subjective smell. Sometimes the term "subjective" is used for the patient's abnormal states of mind, pain, for an example, as when it is said "all his symptoms are subjective," in contrast to the symptoms the medical man can himself testify to, spasm, paralysis, cardiac, murmurs, &c. These different contrasted applications of the two terms lead to great confusion in medical writings, and particularly when the same writer uses them sometimes one way and sometimes another. I imagine even that some philosophical writings are difficult to understand, because the term "subjective" is used sometimes for mind and sometimes for organism; and the term "objective" sometimes for nervous states and sometimes for environment (both the organism and the environment are "outside" mind). Hence, without venturing to say how the two terms ought to be used, I may properly say how I use them in this article. I use them both as psychical terms; the term "subjective" answers to what is physically the effect of the environment on the organism; the term "objective" to what is physically the reacting of the organism on the environment. The reader will note that it is not said that subjective stands for what is on the physical side the organism, and objective for what is environment, but that they are used for what are on the physical side the two conditions of the organism in its correspondence with the environment. The correspondence is duplex, and all mental action is a rhythm of subjective and objective states.

Whether vertigo of the kind defined can be shown to occur most often during discharge of the *left* cerebral hemisphere or not, it is, at any rate, a crude development of the most re-

presentative of all objective sensations. Ocular movements represent the most special adjustments of the organism to—of all its reactions upon—the environment. They are the movements especially concerned during orientation. The vertigo mentioned is a crude sensation occurring during strong discharge of the nervous arrangements for these movements, and is désorientation, as coloured vision is blindness. In fact, the so-called warning (vertigo) is itself consciousness ceasing in a particular way. What is physically a losing of the most special adjustments of the organism to the environment (désorientation) corresponds to what is psychically (object) consciousness ceasing.

Since most people are right-handed, the left cerebral hemisphere in most people represents the most objective movements, those movements for most specially operating on the environment. Moreover, speech is a process by which are symbolised relations of things in, or as if in, the environment[1] or things considered objectively; nearly all evidence goes to show that the left cerebral hemisphere is in most people the one concerned during speech. I make these remarks on speech in order to ask if temporary aphasia does not more often occur after those epileptic seizures which start by vertigo? I am not speaking of the well-marked and very definite temporary aphasia which is found after some right-sided epileptiform seizures. After some epileptic seizures there is temporary "abnormal talking," and this is sometimes aphasic.

Smell and taste are the two of the five special senses which, even popularly speaking, are the most subjective. Besides the five special senses there are sensations which Bain calls "organic" and Lewes "systemic." The common epigastric sensation preluding epileptic attacks is, I suppose, a crude development of the sensation of hunger—the most representative of systemic sensations. If so, the epigastric warning-sensation is (subject) consciousness ceasing. Hunger is, on the one hand, a desire for food, on the other hand it represents the whole of our tissues as to their state of nutrition. At any rate the epigastric sensation is a systemic and subjective sensation.

[1] Of course one does not use the term "environment" in its narrow dictionary meaning. If a man says his leg is longer than his arm, he is as much making a statement of things objective as when he says this stick is longer than that.

I do not make the statements as to the side first in spasm in relation with "subjective" sensation warnings, for any theoretical reason. On the contrary, they are antagonistic to certain conclusions I have come to from a different kind of evidence. It was a matter of surprise to me to find evidence of left-sided spasm at the onset of convulsions with these "warnings." I used to think the more "subjective" of sensations were chiefly represented in the posterior part of the left cerebral hemisphere. The inference that they occur most often in cases when the first spasm is left-sided is from observations, and I say again, observations, all of which cannot be certainly trusted, the reports of patients and their friends not being always to be relied on.

We have also to note the side first in spasm in cases of "warnings" by crude development of the "objective" sensations, sight and hearing. I think it is usually, but certainly not always, the left. Here, again, we must enquire about right and left-handedness. It is certain that in some *epileptiform* seizures with coloured vision the first or sole spasm is right-sided. Alexander Robertson ('Brit. Med. Journal,' April 18, 1874), narrates a case of right-sided (epileptiform) convulsion with red vision. I have recorded the case ('Med. Times and Gazette,' June 6, 1863), of a woman who had right-side beginning seizures, and at other times "attacks" of coloured vision. A left-handed man under my care in the Hospital for the Epileptic and Paralysed had fits beginning by a noise in the *left* ear. There was in that case tumour of the right cerebral hemisphere within the upper temporo-sphenoidal convolution—Ferrier's auditory centre. Gowers examined the specimen for me, and mentions the case in his second Gulstonian Lecture.

All the crude sensations above mentioned are psychical states, as indeed the term "sensation" implies. It is a grievous error to consider sensations as being active states of sensory nerves or sensory centres; doing so leads to taking such warnings as coloured vision to be of the same order as spasm of ocular muscles, or discharge of centres for them; the coloured vision is of the same order as vertigo. When we say that a patient has numbness and spasm of his hand at the onset of an epileptiform

seizure, we are speaking of things different in two ways: the numbness is a crude sensation, a crude psychical state; the spasm is a physical symptom: the former occurs *during* discharge of sensory, the latter *from* discharge of motor elements of the centre. Confusing sensations with states of sensory nerves or centres leads also to ignoring that sensations occur during energising of motor centres. Nor should we use the term "bodily sensation," unless it is understood to mean a sensation referred to some part of the body. All sensations are in our minds, neither in the body nor in the environment; the correlative physical states are in the body. The peasant supposes redness, sweetness, heat, &c., to be in external objects, although, inconsistently, he does not suppose pain to be in the pin which pricks him. The physical changes during sensations are always in our own bodies. The best classification of sensations is into those which are objective—sensations referred to, or as if standing for, things in the environment—and subjective sensations—sensations referred to the body. But this classification, for obvious reasons, is largely arbitrary. For an example, taste serves chiefly subjectively, if it is a question of the agreeable or disagreeable, how the thing affects me; it serves chiefly objectively when it is a question whether this thing is bitterer than that, when it is chiefly a question of relations of things outside me one to another.

We can now consider certain psychical states during the onset of epileptic seizures which are much more elaborate than crude sensations. Once more there are reasons for noting in cases of epilepsy the side first in spasm, and whether the patient be right or left-handed. I speak first of certain highly elaborate mental states, sometimes called "intellectual auræ." I submit that the term "intellectual aura" is not a good one. The state is often like that occasionally experienced by healthy people as a feeling of "reminiscence," that on which Coleridge, Tennyson, Dickens,[1] and many others have written. It is sometimes called "dreamy feelings," or is described as "dreams mixing up with present thoughts," "double consciousness," "feeling

[1] Quotations are given from these authors by Quærens, a medical man, who reports his own case—epileptic attacks beginning by "reminiscence"—in 'The Practitioner,' May, 1870.

of being somewhere else," "as if I went back to all that occurred in my childhood." Sometimes there is a definite elaborate vision. Very often the patient is sure of some thought, but cannot describe it in the least. Dr. Coats, of Glasgow, has reported such a case. The patient may describe the state vaguely as "silly thoughts." These are all voluminous mental states and yet of different kinds; no doubt they ought to be classified, but for my present purpose they may be considered together.

These "dreamy states" mostly occur with the "subjective" warning — sensations, smell, and epigastric sensation, and with the supposed gustatory movements and sometimes with spitting. They cannot be owing to an epileptic discharge. It would be a remarkably well-directed and distributed epileptic discharge which would give rise to the exceedingly compound mental state of being somewhere else. Besides, it must not be forgotten that there very often is along with the dreamy state one of the crude subjective sensations mentioned. It is scarcely likely that one thing, an epileptic discharge, should be the physical condition for a sudden stench in the nose—a crude sensation—and also the physical condition for an infinitely more elaborate psychical state. I submit that the former occurs during the epileptic discharge, and that the latter is owing to but slightly raised activity of healthy nervous arrangements consequent on "loss of control" —possibly of some in the cerebral hemisphere opposite the one, which I believe to be nearly always the right, in which the discharge begins.

The elaborate mental state alluded to occurs sometimes without any crude sensation warning, but in some at least of these cases the first spasm or one-sided affection is left. In one case, with the dreamy state, the patient's head at the onset of every convulsion turned to the right. I was interested in finding that he was a left-handed man. This was the exception proving the rule. In one case, that of Quærens (see footnote, p. 199), in which the patient had a "reminiscence," the only local symptom was in the right hand, and the patient was not a left-handed person; this case is a clear exception.

In some cases with the "dreamy state" the patient spits. In one case beginning by sensation of smell the patient spat out, and had no dreamy state. I suppose the spitting has similar significance to the chewing, &c., movements.

It is, so far as I know, rare for the dreamy state to attend warnings of coloured vision and noises in the ear. I do not know of a clear case of such association. This is the more remarkable as each of these crude objective sensations is sometimes followed by a less elaborate mental state, "seeing faces" and "hearing voices" (words), respectively. "Seeing faces" is a vastly less elaborate state than the dreamy feelings spoken of, such an one as "being somewhere else," although much more elaborate than crude sensations. Coloured vision and "seeing faces" are not likely to be both owing to an epileptic discharge. Both are psychical states, but the latter is far more elaborate.

I have notes of but one case in which the dreamy state occurred with the objective warning of definite vertigo.[1] In but one of all his fits had this patient any sort of warning. The dreamy state attending vertigo in that fit was a feeling of being in some other place, "several places," not any particular place. I use the expression "definite vertigo," for I have known two cases in which with sensation of external objects moving in front of the patients, not to one side, there was the dreamy state; in one (the carpenter's case, *vide infra*) described as "silly thoughts." This patient has the feeling as if objects were coming upon him. I think this sensation, and that of objects appearing to move away, ought to be distinguished from the feeling of objects moving to one side. I have seen a patient in whom, according to his account, a "dreamy" feeling did not occur with his "giddy attacks," but with other attacks beginning by some thoracic or epigastric sensation. So far the case is very striking, and is particularly so as to the occurrence of mouth movements with the seizures in which there was the dreamy state. I admit, however, that there are apparently some discrepancies, to which I shall try

[1] Dr. Joseph Coats has recorded a case, already alluded to ('Brit. Med. Journal,' Nov. 18th, 1876), of an epileptic, each of whose fits, with few exceptions, was preceded by giddiness and a peculiar "thought." "Sometimes the fit only consists of the aura, followed by a peculiar feeling in the abdomen, which passes up to the head and back to the abdomen, when vomiting results."

to give prominence. The patient recorded his own case, which was published with additions, some years ago, by Dr. Weir Mitchell, who kindly advised him to see me. He had attacks of several kinds. Weir Mitchell writes: "The spells of pure giddiness have been frequent of late. They come on suddenly, and there are none of the strange mental conditions which attend the other spells." The giddiness was clearly paroxysmal, and was attended by a feeling of turning to one side. When I saw him, I had no idea of the importance of noting the particular side to which the tendency to turn was, and had paid no particular attention to "dreamy states." The "other spells" began, by what I gathered when I saw the patient, to be an epigastric sensation, but which he, in his printed account, speaks of as "severe oppression across the chest," making it difficult for him to breathe. He states in his account, "I unconsciously move my mouth as if chewing, and sometimes will also grit my teeth." He describes his mental condition as comparable to that of one suddenly awakened out of a sound sleep. "He cannot catch hold of the dream, which seems to be quickly passing from him, and at the same time he cannot yet appreciate the state of unconsciousness into which he has so suddenly awakened." He writes also: "The things around me seem to be moving; and if I am reading, the book will appear to be going from me, when at once I feel as if all must be a dream, though well knowing at the same time it must be reality . . . through it all the fear of some impending catastrophe seems to be hanging over me."

Here we have together epigastric sensation, chewing movements, fear, and dreamy state. But we have to note that there is in these seizures apparent movement of objects; so then it may be taken that this is exceptional. The apparent movement of objects of course implies changes in himself, some ocular movements or discharge of centres for them. It may be said that he had vertigo; yet in what he himself called his giddy fits he had no dreamy state. I did not ascertain whether the movement was of *all* objects from him as it was of the book. When there is apparent alteration of the distance of objects at the onset of epileptic seizures they usually

appear to come nearer or to be larger, but may seem to go further off. It may be mentioned that on awaking from sleep objects appear to be nearer, and as we get fully awake they recede. Such apparent displacements of objects at the onset of epileptic seizures no doubt depend on abnormal states of the accommodative apparatus or their centres (under atropine objects appear smaller or further off; the reverse for calabar bean). This is very different from displacement of objects to one side attended by a feeling of giddiness. Lateral movements of the eyes will have to do with estimation of the figure (superficial extension) of objects, and symbolise tactual movements; accommodation has to do with distance: it symbolises loco-motor movements (of arms as well as loco-motor movements, ordinarily so-called).

I heard of no one-sided affection in the attacks with the dreamy state. It is to be mentioned that he could talk in them. In other attacks, which he calls "paralysing attacks," there was movement of the mouth; but he is then aphasic, and probably the movement is one of spasm. In these attacks his *right* hand becomes fixed across his chest, and he loses "all control over his words." There is no mention of giddiness in these seizures. He adds: "The same feeling of uncertainty usually surrounds me as in the other." If this refers to the dreamy feeling, the case is so far exceptional. He has, however, attacks of severe giddiness without any dreamy sensation.

The day I had written the above I was consulted by a right-handed patient, who had left-hemiplegia since 1875, and afterwards, since 1876, occasional seizures, like those of genuine epilepsy. All his attacks began by the same "dreamy" feeling, as if he were falling down a coal-mine.[1] In this case there was no other premonitory sensation except something referable to the ear. There was not, at the onset, a sensation of smell, nor any epigastric sensation, nor any fear. I positively could not get to know whether or not he had a noise in the ear, or in the head, nor even with certainty that it was a noise at all; for the patient, after describing his sensation as a singing, said it was not a real singing, but rather a "dulness."

[1] He had had nothing to do with coal-mines at any time, and could discover no reason for this particular recurring "dream."

But the interest of the case is that upon a damaged *right* cerebral hemisphere there should ensue seizures beginning with the voluminous mental state mentioned. This patient had slight seizures, and also occasionally severe ones; in the severer ones the spasm preponderated on the left side. I have ('Lancet,' March 11th, 1876) recorded cases in which noise in the ear occurred at the onset of epileptic seizures; in the second case this was followed by hearing "voices," a fact, however, which was not mentioned in that report. I have seen many such cases, but the case of the left hemiplegic patient above referred to is the only one I have seen in which there was a voluminous mental state with a "warning" of noise; if, indeed, there was in that case such warning.

There are other kinds of psychical states in connection with some epileptic paroxysms. It is not uncommon for a patient to have the emotion of terror and to look terrified at the onset of his seizures. When so, there is usually the epigastric sensation with it. It is to be observed that sometimes there is fear without any epigastric sensation,[1] and that sometimes, although very rarely, there is with the epigastric sensation a pleasurable feeling. I never remember hearing a patient mention the antithetical (objective) emotion of anger; but friends of patients sometimes describe a look of indignation. Yet some patients say that at the onset of their seizures they feel they must attack some one, or have a hatred against some person present. As anger is the emotion of combat, this is equivalent to confession of feeling of anger. I do not know anything definite as to crude warning sensation with look of anger, at the onset of epileptic paroxysm. Theoretically, one would expect it to be an objective sensation. I believe that when the emotion of fear occurs at the onset of a paroxysm, with or without the epigastric sensation, the first spasm is on the left. I have, however, one patient whose fits always begin by fear—as if, to use his words, "something had given me a sudden fright"—and whose first spasm is always right-sided. He, however, is a left-handed man (his father is also left-

[1] After some slight paroxysms, beginning either by the epigastric sensation or by fear without it, the patient's bowels are moved: a fact, I submit, of some significance.

handed). This patient has a sensation, not at his epigastrium, but at the middle of his chest. (The case has been referred to, p. 201, as the case of the carpenter.)

It is not uncommon for a patient to have the epigastric sensation, to look frightened, to feel frightened, and to have also the dreamy state. In this regard another thing is to be considered. Some patients act elaborately after their paroxysms. I would ask, "Are the actions after the fit, apparently coloured by fear, actions of escape, when the seizures start by a feeling of fear? Is the same emotion carried out in each stage?"

When there is a dreamy feeling, the "dream" also may influence the post-epileptic actions. In one case actions of "running away" occurred after a fit beginning by sensation of smell, and with a "feeling of being somewhere else." Another patient always used to run away after fits, at the onset of which she had the feeling of "being miles away." (Correspondingly for spectral faces, voices, &c., after coloured vision and noises in the ear?) As with the epigastric sensation and fear there is often also a "dreamy" state, we may have actions in accord with the "dream," and also coloured by fear. I imagine the actions in some cases, wherein there is the dreamy feeling, except, perhaps, that of reminiscence (which is, I presume, nothing other than a revelation of the earlier subconscious part of the normal process of recognition), are more likely to be adjustments to some "ideal" environment; the dream being made up chiefly of long-past experiences; the actions may be very elaborate and yet not purposive-looking as to the present environment.

I would suggest a corresponding series of inquiries as to seizures beginning by objective sensations, and especially by vertigo. "Are the actions after these seizures more seemingly purposive to the patient's present surroundings, and often those of anger?" "Are they the result of an 'unremembered' or vaguely remembered 'dream,' made up chiefly of recent experiences?" The expression "unremembered dream" may seem absurd at first glance; but we must bear in mind that Descartes, Leibnitz, Kant, Jouffroy, and Hamilton believed there to be no dreamless sleep. It may, however, be held that

there is nervous activity without any attendant mental state in deep sleep, that it is really dreamless, and that any "mental modifications" come during waking. Similarly it may be held that when actions after a fit are very purposive-looking there is no "dream," but yet such kind of activity of nervous arrangements as would in imperfect sleep give rise to a dream, made up chiefly of recent experiences.

When actions are always the same in a patient after every fit, it may be that they are in accord with what occurred about the time of his first seizure.

To repeat. I ask, on which side is the first spasm, or other abnormal condition, in cases beginning by crude sensation of smell, by movements of chewing, &c., by the epigastric sensation with or without fear, and in cases where there is fear without any epigastric sensation? I think it is usually the left side. Again I ask, on which side is the first spasm, or other abnormal condition, when there is a "dreamy state" with or without any of the above-mentioned phenomena? I think it is usually the left. We ought to inquire also whether the patient be left or right-handed. Correspondingly for cases in which there is a crude objective sensation as a warning.

I would ask also that the nature of the actions, when they occur after a fit, should be considered (*a*) as being complex or simple; (*b*) as purposive-looking, or seemingly purposeless; (*c*) as unemotional, or as coloured by anger or fear.

I doubt not that there is some order throughout, from the warning to the end of the post-paroxysmal stage. The above imperfect sketch, the best I can do, is the result of much labour, as a contribution towards discovering this order. I am far from asserting that I have done anything of value towards this end, and trust I have fairly acknowledged the difficulties and uncertainties necessary to investigations in which we have to trust so much to our patients.

NOTES ON LEFT-HANDEDNESS.

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MY attention was directed to the subject of left-handedness by a series of articles by Mr. Charles Reade. The view advocated by this writer was that naturally the left hand is as readily used as the right, but that inveterate custom has led men to give an arbitrary preference to the right as the hand which uses the sword, while the left is condemned to bear the shield. He attributes the comparative awkwardness with which the left hand is used to the great care which women take in infancy to accustom children to use the right hand in all difficult operations which require only one arm for their use. Certain it is that infants seem at first to use both hands indiscriminately, and the one arm seems to be at first as well developed as the other, though in process of time the right arm gets more muscular than the left. It is, however, difficult to understand how all nations and tribes, without exception, have in all times of which we know anything given the preference to the right hand, unless they have got some natural reason for so doing, independently of arbitrary usage. Anatomists have pointed out a slight lateral curvature of the vertebral column in the dorsal region, the convexity of which is turned towards the right side, and some have attributed the preference given to the right arm to this bend or curve of the right side. It is, however, more likely the effect than the cause of right-handedness, a deviation caused by muscular action, as explained by Bichat. As most persons are disposed to use the right arm in preference to the left, the body is curved to the left when making efforts, as in pulling, for the purpose of giving an

additional advantage to the muscles, and enabling them to act with more power on the points to which they are attached, and the habitual use of this position gives rise to some degree of permanent curvature. In support of this explanation of the fact, Beclard has stated that he found in one or two individuals who were known to have been left-handed, the convexity of the lateral curve directed to the left side.

On making inquiry in the September of last year, which was done at the request of Dr. Crichton-Browne, who was interested in the subject, I found that there were in the Institution 78 children, 49 males and 29 females, who were using their right hands normally. There were 14 boys and 9 girls, 23 in all, who appeared to use indiscriminately either the right or the left hand; while 7 boys and 6 girls, 13 in all, were decidedly left-handed. On going over our pupils this year, it was found that we had 53 males and 31 females who were right-handed; that 11 boys and 7 girls, 18 in all, used either hand indiscriminately; while 9 boys and 7 girls, 16 in all, were decidedly left-handed. Three girls had ceased to be left-handed during the year, that is, they now use the right hand for sewing and working, where formerly they did the left.

Wishing to ascertain how many left-handed there were among ordinary children, I applied to Mr. Paterson, the teacher of the Sessional School in Larbert, who communicated to me the following observations. Amongst 54 children, whose ages ranged from 4 to 7 years, 8 children had come to school left-handed. They were not allowed to use their left hands in writing or ciphering. Great trouble had, in fact, been taken to make them desist from using their left hands. Some of them had to be kept near the teacher at their writing, but in the playground they threw stones and played at bowls with their left hands. It was thought that left-handedness seemed more prevalent with children likely to be neglected in infancy, but this could not hold good of two of the eight. These children, originally left-handed but now trained to write with the right hand, were not less expert than the rest, and two of them were extra good writers. Of 104 older children out of 52 families 6 boys and 5 girls were left-handed; that is to say, they used the left hand at play, but in the school wrote, or were made to

write, with their right hands. In the whole school, out of 70 girls, 11 girls were left-handed; and out of 88 boys, 8 were left-handed. Thus there was a percentage of 15 girls and 9 boys who were left-handed; and, putting both sexes together, 19 out of 158, that is, 12 per cent. In 1878 the proportion of left-handed amongst our idiots was 11 per cent., somewhat less than in the Sessional School; and in 1879 the proportion was of boys left-handed, 12 per cent.; of girls, 15 per cent.; of boys and girls together, 13 per cent. From this it appears that left-handedness is not more frequent with imbeciles than with normal children. But the cases amongst the imbeciles were much more decided in their left-handedness, generally using their left hands for all purposes; and in the Sessional School the children were all classified as left or right-handed; whereas in the Institution there is a class as large as the left-handed who use both hands indiscriminately. I am aware of the objection to having such an intermediate class, but it was felt impossible fairly to state these children as either right or left-handed.

Thus in the Institution at present, 15 per cent. both of the boys and the girls use their right hand or their left hands indiscriminately. So if there are no more imbecile children decidedly left-handed, there is a much smaller percentage of children who are decidedly right-handed; for while of the normal children in the Sessional School 88 per cent. were right-handed, of the imbecile children now in the Larbert Institution only 72 per cent. were decidedly right-handed.

As idiot children are much less tractable than other children, and it is much more difficult to teach them to use their limbs properly, these results would probably be considered by Mr. Reade as confirming his theory; and it is difficult to deny that custom has a certain power in inducing all those who have a weak tendency to prefer their left hand, to make use of their right one instead. Some people who have been left-handed in infancy, and who through persevering education have been made to use their right hand for acquired work still continue to use their left leg preferably to their right; for example, in kicking, or setting off down a slide.

Luys has asserted that the left side of the brain is earlier developed than the right, so that the left hemisphere in the

new-born child is a few grammes heavier than the right; and in the adult the right hemisphere has been found generally to weigh heavier. If this observation of Luys be correct, the superior weight of the left hemisphere at birth might be held to confirm as well as to account for the habitual preference given to the right limbs.[1] Moreover, if the head be carefully examined, it will be found that its contour is rarely symmetrical on both sides. There is generally a greater protuberance on the left side, a little above or behind the ear, than on the right. On the left side we have a greater curve than on the right, the right side being flatter. This would indicate a greater development of the brain on the left side, near the region where physiologists have placed the motor area of the brain.

In order that there should be less chance of error, I took the cranial outlines of the heads of our inmates with a conformateur. The bulging above or behind the ear in the left side was found to prevail. A very common form of the head was a bulge on the left, behind or above the ear, and a bulge on the right side in front, at the angle of the forehead. Thus the greater size of the left posterior region is made up to a certain degree, by bulging on the right anterior angle. Most of those had a bulge, or increased curvature, on the right side, above or behind the ear; but this did not hold good of all cases.

In a few instances of left-handedness there was very little

[1] Since writing this paper I have had an opportunity of examining two cases which may bear on the subject. B. N., 11 years old, epileptic imbecile, left-handed from early infancy, and subject to very frequent fits, which are occasionally on right side only, when he retains consciousness and is able to answer questions. About 8 months before death he had these partial fits, followed by temporary paralysis of right arm, which also implicated the face. Died apparently from exhaustion, following repeated general convulsions. Whole surface of cerebrum deeply injected, bright red, with patches of deeper hue. The left side of encephalon was smaller than the right. This was especially noticeable in the middle lobe, viewed from below, and the middle fossa of skull was smaller. The pons was also smaller on the left side. The right side of encephalon weighed $20\frac{3}{4}$ oz.; the left side $15\frac{1}{2}$ oz. H. M., paralytic imbecile, had a second shock after coming to institution, aggravating the old paralysis of the right side. Lingered on for a year in great debility, sinking into deep dementia. The cortex of cerebrum was much diseased and wasted, with adherent membranes and accumulation of fluid within the ventricles. The right side of brain weighed $16\frac{1}{4}$ oz.; the left weighed 15 oz.

difference, in others there was actually a bulging on the left side. Putting aside the cranial outlines of those, the surface of whose heads were bigger on the right side, I found that 7 were decidedly left-handed, 7 belonged to the indiscriminate class, and 10 were right-handed. Of those whose outline seemed larger on the left side, 5 were decidedly left-handed, 8 indiscriminate, and 50 right-handed. One girl, who was very decidedly left-handed, was much bigger on the left side. In one boy, almost a mute, but with so much intelligence that he might be styled an aphasic imbecile, and who was left-handed, the greater size of the left side of the head was very decided. Those who are left-handed generally kick with the left foot, but not always. I have the cranial outline of a man who was left-handed in childhood, but who was trained to work at a trade with his right hand. In his case the bulging in the head is on the *left* side. On taking the cranial outline of a friend, I remarked that he ought, from the contour of his head, to be left-handed. He answered that he could use both hands almost equally well, and, as a proof of it, wrote his name upon the profile card with his left hand. He observed that his father had been left-handed, and I have met with other facts which make me think that it is worth while inquiring whether this peculiarity may not be hereditary.

It is but fair to acknowledge that I am indebted to Mr. Crochley Clapham for the idea of using the conformateur in this line of inquiry. This able anthropologist has stated, as the result of his extensive inquiries,[1] "that the most common form of skull met with was that having its greatest transverse diameter posterior to the median line, and being most protuberant, with reference to the long diameter, on the left side. This condition of left-handedness was present in 81·771 per cent. of the insane; but, as shown in the regional development table, was much more marked in some diseases than in others; and, as bearing upon the question of which is the 'driving side' for convulsions, it may be interesting to notice that left-handedness is not only very common in epilepsy, but is also

[1] See the paper on "The Cranial Outline of the Insane and Criminal," by Crochley Clapham, L.R.C P., &c., and Henry Clarke, L.R.C.P., &c., in the 'West Riding Asylum Reports,' vol. vi. p. 154.

more pronounced in this disease than in any other form of mental affection." Mr. Clapham also observes that the "numerical superiority of the left-'headed' agrees with observations made by numerous writers on the greater relative weight of the left cerebral hemisphere. That it has an obvious, though by no means exclusive, connection with right-handedness is shown on comparing the criminal tables, where the opposite state of right-headedness was exhibited in a number of left-handed individuals." Some may hold that this one-sided development of the brain is really the consequence not the cause of right-handedness, that just as the right arm becomes stronger and more muscular than the left by constant use, the corresponding portion of the brain also takes an increased development.

The view that the habitual preference of the right arm really arises from a greater or earlier innate power in the left hemisphere, seems strengthened by the well-known facts of aphasia, and the physiological experiments confirming and illustrating them.

It is scarcely needful to recall that on the left side of the brain in the lower part of the third frontal convolution, or in the operculum, is situated what has been called the speech-centre, where motor acts of articulation take their origin. On the destruction of these parts on the left side, the power of imitating words is lost, though the aphasic patient generally understands words. As the utterance of words requires the combined action of both right and left muscles of the vocal apparatus, it is clear we have here a fair case of one-sidedness in the hemispheres, which can only be denied by denying that in *most* cases of aphasia the lesion is on the left side, and that it is often associated with paralysis of the right arm. This left-sidedness, at any rate, is beyond the reach of mothers and teachers.

Dr. Ferrier observes, in his book on the 'Functions of the Brain':[1] "As regards the articulating centres, the rule seems to be that they are educated, and become the organic seat of volitional acquisitions on the same side as the manual centres. Hence, as most people are right-handed, the education of the centres of volitional movements takes place in the left hemi-

[1] Page 278.

sphere. This is borne out in a striking manner by the occurrence of cases of aphasia with left hemiplegia in left-handed people. Several cases of this kind have now been put on record."

An instance is given by Dr. Hughlings-Jackson,[1] who adds :—"It is admitted that these are cases of left hemiplegia with aphasia in persons who are not left-handed." In such cases we must suppose the main articulating centre to have been in the right hemisphere without inducing the motor centre of the left arm to take the lead. This seems to prove that the connection of sequence or concomitance between the education of the centres of articulation and manual execution in the same side of the brain is at least not inseparable. As a rule the left hemisphere seems functionally the most active, though the right side has also its work, and the left hand executes important actions, such as holding the reins on horseback.

Mr. Crochley Clapham has noticed that left-handedness is more marked in men than in women, which "goes to substantiate the 'crossed-action' theory, as most female employments necessitate the pretty even use of the two hands."

Dr. Wilbur, of Syracuse, New York, had a friend who professed to be able to distinguish a left-handed man from a right-handed, by attentively watching him without seeing him make any special use of the arm. He grounded his observations on the premise that the appearance of the face was also different in the left-handed. Certainly, the configuration of the face and the fulness of the facial muscles often vary a little on each side; but, as far as my observations go, I am not prepared to undertake the challenge sustained by Dr. Wilbur's friends.

Trousseau[2] has remarked that the right and left sides are subject to different diseases. Neuralgia is so much commoner on the left side, that for three years during which he kept notes he did not observe a single example of it on the right side of the chest, when real neuralgia was carefully distinguished from pleurodynia, pleuritic stitches, and hepatic colic.

[1] 'Brain,' October, 1879, p. 329.

[2] 'Clinique Médicale,' Paris, 1868, vol. ii. p. 668.

It may be asked, that where left-handedness has shown itself in a decided manner, is it proper that so much trouble should be taken to make the child break it off? By so doing it is evident that the teacher gives the child a great deal of trouble and perplexity. A left-handed child forced to write with his right hand through fear of punishment, is very much in the same condition as a right-handed one who should be forced to hold his pen in his left. There is no proof that people who remain decidedly left-handed all their lives are less skilful with their hands than others. One of the earliest mention of left-handedness is in the book of Judges, where it is recorded that amongst the 26,700 fighting men of the tribe of Benjamin, there were 700 chosen men left-handed; every one could sling stones at a hair-breadth, and not miss.

On the other hand, it may be argued that as those who are originally left-handed generally retain the use of their left hands for a number of offices, by teaching them to use their right hand we are to a certain extent making them ambidextrous. Another consideration seems much less capable of dispute; since custom has clearly so much play in determining what hand shall be used, it is a misfortune when our acquired expertness becomes the exclusive property of one hand. We ought to practise the left hand as well as the right in difficult manœuvres. There is no doubt, for example, that it is of great advantage for a surgeon to be skilful in the use of both hands, and this can be only obtained by practice commenced in early years.

Critical Digests and Notices of Books.

The Brain as an Organ of Mind. By H. CHARLTON BASTIAN, M.D., F.R.S., &c. Kegan Paul & Co., London. International Series, 1880.

THE taking title of this book at once suggests the question or questions, what is the brain? what is mind? and what is an organ? and if they do not meet with a clear response, it is not from any want of inclination or ability on the part of the author to deal with them in the fullest manner which scientific knowledge will permit. Dialectic skill, the heritage of metaphysicians, is more and more shared by these new rivals, who are more formidable than the older antagonists the pure physicists; rivals whose forcible endeavour is to prove the correlation of all knowledge, and in this domain especially to correlate those ancient foes, metaphysics and physiology. Surely nothing in these days can be more narrow and misleading than for the metaphysicians to exclaim, "Oh, that is physiology, we have nought to do with that," unless it be a like response from the physiologist in his ignorant abuse of the methods of metaphysics. It is no wonder that the elder science, which attained to the perfection of Aristotle and Plato when real knowledge of living organisms was barely commenced, should have stood alone in sulky dignity long after it ought to have welcomed the alliance of the new power. Nor is it inexplicable that physiologists should tend towards exclusiveness in their attempts to answer all the problems of life by their own methods. But now that the sciences are continually being differentiated and multiplied does it become the more needful to appreciate their essential correlation; and in none so much as in the sciences of mind, and that science of living organisms without which mind, so far as we know, cannot exist.

And when we speak of these sciences of mind, which developed into activity long before a nerve-cell or tube had ever been seen or imagined, we mean, not simply the abstract sciences of ethics or metaphysics, or that guess-work of immaterial ontology which it may still amuse some to maintain and some to decry with equal impotence and futility, but of those concrete sciences of poetry and history, and all those *literæ humaniores* which teach us so much of what we know of the motives and actions of man, and without which the knowledge of the whole mass of physical science would be poor indeed. Conceive the great Chinese nation having got the start of all the world by a thousand years in the art and science of physiology, as they did in those of the fictile manufactures! Clearly they might have attained to a knowledge which we can scarcely imagine of the physical processes of sensation, emotion, and memory, while they remained the children they are in the real science of mind and motive. And this is what our physiologists might do, if their labours did not constantly fecundate other knowledge, which again gratefully repays its debts in adding force and breadth to their own powers in their own domains. Dr. Bastian is an excellent example of this modern combination of the dialectician with the physicist, and his book is a good instance of the correlation of metaphysics with physiology—leaning, it is true, to the latter with a heavy bias, but frankly accepting the more abstract science in all except its unintelligible and indeed inconceivable ontology. It is somewhat intriguing to one's curiosity that writers like Bastian and Bain should think it worth their while to continue these arguments against an ontological essence of mind, while the very idea of such a thing remains unpresentable to the mind, and when it seems to be certain that belief in such a thing is belief in a verbal formula, and not in a real idea. Dr. Bastian commences with an interesting chapter on the Uses and Origin of a Nervous System. As might be expected from his well-known views on the beginnings of life, this chapter contains opinions which are not generally accepted. Dr. Bastian assumes that it is "commonly admitted that many of the lowest forms of life cannot positively be assigned either to the vegetable or the animal kingdom," "creatures of circumstance,"

among whom "variability reigns supreme," "appearing now as animals, now as plants," which he designates "ephemeromorphs." Modes of growth, he says, have been observed to occur in them with frequent and rapid changes from vegetable to animal, and from animal to vegetable; and he compares these changes to the well-known metamorphoses of form and nature amongst simpler kinds of matter, as in the changes from the crystalloid to the colloid form of molecular aggregation demonstrated by Graham, and to the interchangeable states of carbon, phosphorus and sulphur. While we take the author's word for the peculiar qualities of his ephemeromorphs, the analogy to the allotropism of inorganic elements seems forced, and the manner in which these considerations lead through the movements of plants to those of animals, and to the differentiation of nerve tissue, is not very obvious or convincing. No doubt it is right to begin at the beginning, and to seize the first link of the chain if we can, but many physicists will think that there are some important links wanting between allotropic sulphur and the Amœbæ—"the simplest types of unquestionable animal life" with "high inherent activity," and with "the power of executing well-marked independent movements, and of feeding upon solid food." Most people will even think that there is an important link wanting between the allotropic chemical and the self-multiplying Confervæ, and another between them and the amœba, with its high *inherent* activities and *independent* movements.

With independent movements and "the selective power which the amœba seems to manifest," we first appear really to touch upon the problem of mind, for we are supposed to be within the confines of science, so that any misgivings as to the mind of the inorganic universe, or even the loves of the plants, must be excluded. But what do the independent movements of the amœba in the selection of food mean? Probably the author will concede that in a certain sense no movements even the most voluntary of the highest known organism are really independent; for whether the will be determined or not, the movements it ordains are determined, and are therefore not independent. But in a milder sense the movements of the amœba may well be supposed to be far less dependent upon

outward circumstance than those of the insectivorous plants, and, indeed, to indicate the high inherent activities here attributed to them. And what can the inherent conditions be in which these inherent activities develope unless it be the first dim glimmering of consciousness, a condition analogous, if not homologous, to our own consciousness? That no one can positively affirm this to be so or not to be so, is as true of the amœba as of the crayfish, of which Huxley rightly says that without being a crayfish, or at least having been a crayfish and remembering, no one can say whether it is conscious or not. But the same may be said of the author himself. One can only draw the "legitimate inference" that he is conscious. One can by no means claim any positive knowledge that he is conscious. This, however, also would appear to be a legitimate inference, that when an organism, however lowly, shows by independent movements in the selection of food that it possesses inherent activities, it is probable that it distinguishes between the being which selects and the food selected; that is to say, between the *me* and the environment, the *ego* and the *non-ego;* and if this be not consciousness, we can form no conception of what it is, or of what rudimentary consciousness itself may be. This inference, which to many may appear forced and unwarranted, the author does not draw, however near the quoted terms he has employed may seem to lead to it. For ourselves we abide by it as a legitimate inference from the continuous chain of organisms in which there appears to be no break in consciousness. And if it be said that in the amœba and other of the very simplest animal organisms there is no separate organ of consciousness, it may be replied that we have no proof that such a separate organ is necessary for the purpose; and for that matter that the amœba may as well be considered a nerve-cell with powers of digestion as a digestive cell with nervous function. Our author's views as to the states of consciousness of the lower animals are much more restricted. He thinks that even the opinion of G. H. Lewes, that all nerve centres are the seats of nervous sensibility, "would only lead to its extension, and soon make it almost impossible to deny a similar attribute to the leaves of the sun-dew and other sensitive plants, or indeed to stop even

here. Endless confusion might thus be produced without commensurate gain." But no one can believe that any plant is really possessed of sensation, although called sensitive; and the motive of immediate profit or loss to science ought never to be considered, for nature gives long credit to drafts upon her resources which may at first seem untrustworthy. When Dr. Bastian states that "the very existence in the lower animals of any conscious states analogous to those which we ourselves experience is a matter only of warranted inference," he is stating the question with philosophical accuracy, which is more than is needful in a discussion as to the amount of scientific probability. Whether or not sensations or impressions, being "associated with certain subjective phases answering to what we call states of consciousness," arise only "at a certain stage in the complication of the nervous actions of the lower organisms," when the "ingoing molecular movements traverse nerve fibres, and thence diffuse themselves among related groups of nerve-cells," as the author thinks, or whether, as we think, there is an uninterrupted downward chain from man to the amœba, in which states of consciousness, gradually receding from the highest mind to the dimmest differentiation between self and not-self, is a question only of more or less legitimate inference and probability. The hypothesis of continuity, however, does seem more in accordance with the ways of nature and the facts of the case than of discontinuity, which seems to require that "the quick succession of changes in a ganglion, implying as it does perpetual experiences of differences and likenesses, constitutes the raw material of consciousness." We must pass by the excellent anatomical chapters on the structure of the nervous systems and the brains of the different classes of animals, to arrive at the important chapter on the Scope of Mind. We may very well omit as superfluous in these pages any notice of the author's well-put objections to the custom of speaking of mind as "an actual independent existence," or even as "a definite self-existing principle." Mind is not this or that, but according to John Stuart Mill, "what consciousness directly reveals, together with what can legitimately be inferred from its revelations, compose by universal admission all that we know of mind, or indeed of any

other thing." And this passage from the great logician seems to supply the author with a satisfactory account of what mind is, subject, as we shall see, to certain extensions which he himself proposes; and he follows up the curious distinction he makes between the direct revelations of consciousness and the inferences we draw from them, with the reflection that it would be an absurdity to rest content with the conclusion, that we must lean implicitly and conclusively upon these direct revelations of consciousness. But the author does not inform us where inferences exist if not in the consciousness, unless he intends to include them among those "mental phenomena" with which he contrasts "any *other* natural phenomena," because "all other phenomena can only be known in terms of mind." We do not know that this application of the term phenomena is quite new, but it certainly is not quite accurate, for how can a subjective state by any possibility be an appearance? "The imaginative embodiment of these subjective states into a non-corporeal or spiritual Ego, is not altogether (more) surprising." But our author here again permits himself to contemplate, and indeed to be a little scared by the supposed consequences of the truth he is seeking. "If we were to lean implicitly and exclusively upon these direct revelations of consciousness, we must, as the history of philosophy has shown, inevitably commit ourselves to a system of universal scepticism, needing, as Hume proclaimed, a rejection of all grounds of certainty for our belief in an external world, in body, and, indeed, in mind, as an entity—leaving to each one of us a mere fleeting series of conscious states as representatives of the totality of existence. The absurdity of resting content with such a conclusion has been commonly recognized both by philosophers and mankind in general. In fact we use our consciousness to enable us, in imagination at least, to transcend these direct revelations of consciousness. They are by each one of us invariably supplemented and modified, where necessary, by what we deem to be legitimate inferences. Our actual present knowledge is made up of a closely interwoven, potential, but intelligible fabric derived from actually existent, from remembered, described, or inferred conscious states or relations between them, together with inextricably intermixed, and

more or less legitimate inferences therefrom." Thus we have "inferred Conscious States" and "legitimate inferences therefrom" which are not Conscious States, in order that we may not be too sceptical, and reject grounds of certainty, and we so, in imagination at least, transcend the revelations of Consciousness. Surely also "endless confusion may thus be produced without commensurate gain;" and surely in our search for knowledge as to the nature of mind, the bogie of scepticism regarding the existence of body need not scare us, as it certainly will not prevent us from eating our dinner.

But we shall never get to the end of our task if we notice all the author's opinions, which are suggestive, and which seem to call for examination if not criticism, that we may see in what sense or to what extent we may accept them; for this work is far removed from the trite methods of those imitative minds which conceal a rickety knowledge of physiology with a poor veneer of mental science. No doubt it is very difficult to amalgamate physiology and psychology, or to bring into unison what our author calls the subjective phenomena and the objective phenomena of thought, and the difficulty is increased by the fact that the popular if not also the scientific terminology of the matters in question is mostly inherited from times antecedent to scientific inquiry. And this leads us to remark that however we may be inclined to concede that "the notion that Brain is the exclusive organ of Mind can no longer be entertained," and however we may sympathize with the author in feeling the difficulty which arises from excluding unconscious states leading to or linked with conscious states from the term Mind, still we cannot think that "this is no question of choice, but one of absolute necessity. The meaning of the word Mind must be very considerably enlarged, so as to enable us to comprise under its new and more ample signification the results of all the nerve actions, other than those of outgoing currents." On the contrary we think that something else may be done, and certainly must be done, in the gradual formation of a new terminology more consistent with the development of neurological science; and perchance in this manner, even the present generation may escape from being called upon "to bear the discomfort and inconvenience arising from an altered meaning of

the term Mind"—a duty of sufferance which the present generation would probably repudiate. In this manner those who do wish to think and to communicate their thoughts accurately would gradually provide themselves with a verbal armament as suitable to their purpose as their knowledge would enable them to fabricate. And who knows what changes in scientific terms may be found needful, while leaving the old landmarks of language where they are? Nerve centres, discharges, currents, reflections, inhibitions, &c., are they not all terms based upon hypotheses more or less probable but as yet unproved—all of them terms which new knowledge of neuration may at any time relegate to the limbo where *phlogiston*, *humidum radicale*, and other old verbal implements which rust in disuse and neglect?

The lucid chapter on Sensation, Idealism, and Perception concludes with a pregnant paragraph on "the possibilities of intellectual affection and action bequeathed to an organism in the already elaborated nervous system which it inherits. Within this nervous system lie latent the creature's forms of Intuition or forms of Thought, which need only the coming of appropriate stimuli to raise them into harmonious action. It is the fact of the previous orderly organization of the structural correlatives of mental processes, which causes some degree of those modes of mental affection, known to us as Feeling, Intellectual Action, Emotion, and Volition, to be engendered even in the young untaught organism, in response to suitable stimuli."

This explanation of what may be called the mental Instincts, the cause of untrained and untaught cerebral action in the young, is surely also the largest part of the answer to the question of those neurations in adult lower animals which are more commonly known as Instincts. This, indeed, is admitted in the conclusion of the interesting chapter on Instinct, its Nature and Origin, in which the author says: "There can be no doubt that if our means of knowledge were greater than it is, we should be able to explain these and all other Instincts by reference to the doctrines of inherited acquisition, and natural selection, either singly or in combination." It seems to us, however, somewhat unnecessary to eliminate these two doctrines out of one process; for survival of the fittest can

only become influential through long-continued inheritance. We must pass by the excellent chapters on the Brains of the Quadrumana, the Mental Powers of the Higher Brutes, the Configuration, Structure, Function, Relations, &c., of the Human Brain, that we may glance at the chapter From Brute to Human Intelligence. In this linking chapter the author very fairly traces the influence of the possession of articulate speech, "an endowment which, when once started, —whether by some hidden and unknown process of natural development, or as a still more occult God-sent gift to man—was by its very nature almost certain to have led its possessors along an upward path of cerebral development." Is there not somewhat of a circle, or at least an eddy, in the current of this argument? Speech developes the brain; but does not the possession of a large brain develope speech? As the author mentions, the men of the Post-Tertiary Drift lived for untold ages in a state of extreme simplicity. It is doubtful to what extent they were social or solitary; and we have every reason to believe that their language, if they had any, was most rudimentary. Of course, social habits once attained and language acquired, the progress from hieroglyphics to writing and printing and stenography and telegraphy must have run parallel to a progress from simple concepts not unlike the visual images of brutes, through conceptions more and more general and abstract and complicated, up to the highest development of man's intelligence. The well-considered digest of our knowledge on the Functional Relations of the Principal Parts of the Brain has a chapter separated from that which clearly belongs to the subject, namely, Phrenology, Old and New. In considering this latter division of brain-function, the author does well to remind us that "only within the last century has the great bulk of our present knowledge with regard to it [the structure and functions of the brain] gradually taken shape from amidst the clouds of error with which the opinions of the ancients and the mere speculations of the anatomists of later centuries had enshrouded it;" and he gives an excellent résumé of these opinions, culled and condensed, as he says, for the most part from the writings of Prochaska—that neurological mine which has too often been worked without acknowledg-

ment. The old system of Phrenology of Gall and Spurzheim, founded, as the author says, upon a crude psychological analysis, well capped by a very simple mode of hap-hazard and tentative observation, and fallacious in almost every respect, was very different to the new method of experimentation by which questions of the localization of cerebral formation are carefully put to nature. The yea or nay of the general question of localization of function has underlying it the secondary one whether mental faculties are dependent upon separate areas of brain-substance, or whether cells and fibres, so far as position is concerned, are interblended with others having different functions. "Have we to do with topographically separate areas of brain-tissue, or merely with distinct cell and fibre mechanisms, existing in a more or less diffuse and mutually interblended manner?"

The search for perceptive centres, taken up by Ferrier in "a thoroughly systematic manner," and pursued by him "with characteristic energy," led to results which "deserve to be most carefully studied." The reader is referred to the original for full details of these experiments, "made with much skill and judgment;" the author's opinion respecting them being, that "although Ferrier's determinations of the sites which are of most importance for each sense require more confirmation by other workers than they have yet received, before they can finally be accepted as correct, the discrimination and ability with which his experiments have been conducted should ensure for them that careful and thorough testing which their importance deserves." We have only space left to indicate the great interest and importance of the concluding chapters on Speaking, Reading, and Writing, as mental and physiological processes; on the Cerebral Relations of Speech and Thought; and on Problems in regard to Localization of Higher Cerebral Functions; and to take leave for the present of this most valuable addition to the popular series of science treatises, of which it is the youngest recruit, with the expression of our own opinion that the diverse, complicated, and profound problems of psychology and neurology with which it is engaged have never before been so fully, ably, and lucidly presented to the general reader.

JOHN CHARLES BUCKNILL.

Mind in the Lower Animals, in Health and Disease. By W. LAUDER LINDSAY, M.D. 2 vols. C. Kegan Paul & Co., London, 1879.

THIS is a work of formidable dimensions, consisting of two volumes of more than 500 pages each, the first being devoted to the mind of the lower animals in health, the second in disease. The author tells us that he had at first intended to produce a book of much less size, but that on attempting it he found the materials so swollen that he could not compress it within its present limits, though we confess to the opinion that some diminution of its contents would enhance the value of the whole.

In substance it is composed of a vast quantity of notes, slowly accumulated by the author, gleaned from a wide area of experience covered by travel and reading, while prompted by the motives of a "physician-naturalist," as he styles himself, in the former capacity being specially occupied with mental disease; but neither does there appear to have been enough partiality in the collection, nor has the matter received sufficient elaboration in passing through the author's mind to have exchanged much of its crude note-like form, for the more compact and comely presentment of ingenious theory, although it has been somewhat arranged and adorned with comments.

Dr. Lindsay says (Introd. p. xiv.) he "attempts to *outline* the subject of *Mind in the Lower Animals,* to illustrate their possession of the *higher mental faculties as they occur in man,* of *reason* as distinguished from mere *instinct.*" He is modest in his claims, and intends only a popular treatise, therefore he must not be regarded too harshly.

There is too little coherence in the book to make it an easy task to criticise it as a whole, and the bulk is too great to allow detailed examination. Yet it is soon visible, on perusing the book, that the author's aim is to exalt animals at the expense of man, in opposition to the depreciation with which, by what the author considers human prejudice, they are generally received; though, of course, he does not quarrel with the vast superiority of man in his most complete develop-

ment, he selects the more backward types of the human race —e.g. savages, criminals (whose brutal habits afford the opportunity for sarcasm at our so-called civilisation), idiots, lunatics, &c., for description—to compare with the best examples of the lower animals. Notwithstanding the care with which this comparison is in many respects pursued—and no one can doubt the honesty of the author, though, perhaps, his enthusiasm may be a little mis-directed—there seems a good deal of the art of the advocate in place of the strict impartiality of an inquirer after truth; there is the truth, but not the whole truth. The method and plan employed are, unfortunately, by no means innocent of logical faults; one of the most serious being too common in the scientific examination of a system—such as the mind—of instituting a comparison piecemeal, of each pair of elements in isolation, apart from their relation to the rest of the system. Thus the structure formed by the parts in combination, which is really almost the sole object of search, eludes observation, just as the soul has evaded the anatomist's knife; so that although it might be proved that in certain single characteristics or aspects the excellence might belong to the member of the more humble class, yet it by no means follows that the total mind of the animal in question will equally bear the palm. Scarcely any attention is paid to the important fact that man possesses a larger number of highly-varied and nearly always more highly-developed faculties, and a versatility in their application that is peculiar to himself. Further, the reader feels oppressed by a strained analogy throughout the book—what is outwardly similar being regarded as essentially identical—perhaps to meet preconceived views as to the resemblance, the chief form this takes being the fallacy of anthropomorphism, animals being credited with human motives, on which subject he remarks (Vol. I. chap. xviii.) that interpretation of motives is very difficult, even in our fellow-men; and he assumes that the "attribution of human motives is both legitimate and necessary" (p. 339). As an example of this we would refer to the case (Vol. II. p. 301) of sporting dogs being described as filled with disgust and contempt, and consequently deserting their master, because of his incompetence

and want of skill as a sportsman; but is it not much more likely that their desertion was due to disappointment at failing to obtain the pleasures of a successful chase? If all such interpretations were admissible, one would be almost persuaded of the possible truth of Æsop's fables! Another frequently-repeated variety of the same error is the confusion of similarities of analogy with those of homology, as well as different stages in the development of a function with one another, for in psychology, not only the quality of a particular sentiment, but also the range of its application—i.e. its intellectual equivalent—must be taken into consideration in estimating its real character. Again, he is apt to confound mere imitativeness with spontaneous efforts at construction, thus assigning too much credit to the imitator; also he does not always appear to attach sufficient importance to the power to adapt means to ends, or to distinguish accurately between the qualities learnt by natural experience and those taught by man.

The work cannot claim pretensions to be a system of comparative psychology, for there is not much philosophical comprehension of a standard of comparison, or a thorough scientific analysis of the mind into its several faculties, to be traced in their grades of development through the several classes of the animal kingdom.

The author discards—perhaps with good reason—any classification of mental faculties at present in vogue, but acknowledges his inability to frame a better.

Instead of this, groups of mental phenomena are taken somewhat promiscuously, according to their outwardly prominent features, to head the different sections and chapters of the book; and these groups are sub-divided arbitrarily, like a catalogue, into various lists of minor qualities, arranged, in rather monotonous series, too much like the objects collected in an ordinary museum. This arbitrary arrangement of so large a quantity of facts is certainly one of the chief obstacles to appreciation of the author's work, for it renders the book somewhat dreary to read, though perhaps it may be in accordance with the Baconian rules for conducting the investigation of nature. It might also be objected with some force that there is as yet insufficient material to pursue a

satisfactory comparison between animal and human minds. The resemblance, here performed somewhat cumbrously, is in tone much like the masterly account given on a smaller scale, with much more justice and eloquence, in Darwin's 'Descent of Man.' To have drawn an adequate picture with predominance of the distinctions above the general agreement would indeed have been invaluable, but, as the author regrets, almost impracticable. We think that failure thoroughly to appreciate the points of difference has led him to exaggerate the fundamental identity.

In introducing the work to his readers, the author describes the method he has endeavoured to employ in its preparation, laying down at length what he considers to be the best rules for conducting scientific research; giving due notice of the chief fallacies to be avoided; so that we presume he assails his task, thus guided and protected, with no want of self-confidence. He says he aims only at laying down facts, but is careful to attest them by reference to and examination of the authorities whence he draws them. We do not feel altogether satisfied with either of these attempts, for such a huge record of facts is greatly diminished in value by being imperfectly digested; and certainly many of them demand a heavy tax on our credulity to obtain acceptance, e.g. that sperm whales communicate with each other at the distance of six or seven miles! (Vol. I. p. 420) although the author declares he has been at pains to verify all his borrowed information. We suppose that it must be from a slip of which the author was unaware that we find the absurdity (Vol. I. p. 420) that, among other animals, the *guinea-pig* uses its tail as a respirator, and to maintain its warmth! The chief sources of the author's information are the works of J. G. Wood, Houzeau, and Pierquin, the Animal World, and current newspapers, besides original observations, for which his extensive travels have given him the opportunity.

In spite of the abundance of matter, we are inclined to believe that more profound studies of fewer cases, and the detailed discussion of intricate, knotty, and interesting problems would have been more profitable than the mere deposition and enumeration of the mass of facts which overcrowd

these volumes; it must remain a work for consultation rather than for ordinary reading, though not exhaustive enough to constitute a standard work of reference; for we believe that contributions to science—at least in the inexact department of natural history—lose much of their worth by extreme dryness of statement, just as jewels are less esteemed when reduced to powder instead of remaining of larger size, and being duly cut and advantageously set. However, we will gratefully offer at least one fragment of commendation—to the attempt to give an account of the phenomena of mind without constantly referring to unmeaning nerve-cells, &c., for explanation. The first section consists of a brief outline of comparative psychology, showing the increasing mental complexity in the ascent through the invertebrate and vertebrate classes, although the psychological and zoological scales do not coincide. A chapter is given to show how incorrect is the character given to some species of animals by popular estimation; usually worse than they deserve, though sometimes they make more blunders than are expected of them. Chap. ix. contains a list of alleged psychical differences between man and animals; but they are all held to be neither invariably present in man, nor always absent from animals. The popular belief in a sharp demarcation between instinct and reason is strongly contested; every animal is further said to possess a distinct individuality. Some curious familiar subjects are dealt with in chap. xiii., under the head of "Unsolved Problems," including migrations, &c.; but not a very great amount of the light of reason is brought to bear upon it. Then begins the detailed study of the mind in its normal manifestations: the first subject treated of being the moral sense, and in order to draw as near as possible a comparison between its developments in man and animals, its presence in some lower specimens of humanity is at first considered; it is declared to be occasionally absent, perverted in disease, and to decay in old age. A list is given of a large number of features of the moral sense in savages, &c., of many immoral characters present instead. From man, the step is taken to lower animals; but it would have been well to stay to ascertain how far the terms "right" and "wrong" are at all applicable to animals, seeing that the courses of action

among which they have to choose are less numerous and less embarrassing than in the case of man, and especially as the social conditions of the latter give quite a novel direction to his life. The author admits that "what is popularly spoken of as a sense of right and wrong, of legality or illegality, in the lower animals, may, or will, if strictly analysed, be reduced to a distinction between what is *forbidden* and what is *permitted* by man, who is recognised as a sufficient lawgiver and administrator—what will bring *punishment* on the one hand and *reward* on the other" (p. 181). "But," he adds, "this is just the kind of feeling that exists in the savage, the civilised child, the lunatic, or idiot." But he certainly does not show that it becomes so large a portion of the mind, its pursuit so independent, in any animal as in man; he holds that there is no reality in the objection that what are apparently moral acts are not really so. As corroborative of his views of the moral responsibility of animals, he mentions that their acts were formerly so regarded in courts of law; and in extenuation of their would-be faults, he holds that many of them are due to man's ill-training. The same subject is also treated of in other parts of the work, viz. chap. xxii. on Law and Punishment, where it is stated that some animals have rules laid down by authority and custom—a law of right as well as a law of might; they have rights in property and rank. Some birds have apparent judicial proceedings; and there are exhibited a variety of punishments. The obverse of the same question is handled in Vol. II. chap. xi., on Crime and Criminality, showing that they may be guilty of offences against man's or their own laws; their faults being theft and destructiveness, including murder and mutilation, and they are conscious of their criminality. In a chapter on "Sensitiveness" is shown their keen susceptibility to the opinions of their fellows, &c. The ethics of vivisection are not discussed. Next comes an account of the religious feeling—first in man, in whom it is shown that there is sometimes no notion of any Supreme Being; in savages there is fetish and ancestor worship, and hero-worship even in civilised man. Then the religious feeling is stated to exist in many animals; but the proofs of exalted feeling seem scarcely convincing. That many domestic

animals look on man as a god is very likely true; yet, in comparing them with men, it must be recollected how much more easy it is for them to form such an idea, having the object of reverence within reach of their perception. The analogy is surely carried much too far in ascribing attendance at church by dogs to gratification of a religious sentiment! and piety to parrots for repeating prayers!

Next, the capacity for education is discussed, and Ch. X. contains some interesting information of the training required by the young of many species to develope some of even the strongest instinctive aptitudes.

Next the distribution of language is considered. We cannot understand on what grounds it can be held that "animal language is more comprehensive than, and quite as eloquent as, man's." Houzeau's remark also is quoted with approbation, that the language of certain savages is inferior to that of various animals. The author expresses an opinion that a professorship of comparative language should exist in all universities, and due attention given to all forms of expression common to other animals with man. Several pages are occupied with a long alphabetical list of the significations of animal vocal sounds; and it is noteworthy that all are names of states of feeling, thus suggestive of the interjectional origin of language, and of the degree to which the mind must have developed before articulate speech arose. Laughter is shown to be neither universal in, nor peculiar to, man; weeping is an expression of grief common to many animals.

The rest of Vol. I. is devoted to more special phenomena; under the heading of "Adaptiveness," it is shown of what variation the powers of animals admit; what use they make of the members of their own bodies, of natural objects, and man's instruments; that they occasionally delight to clothe themselves, prepare food, and provide themselves with shelter; also that they combine to form societies, with one or other form of government; that they often show considerable acquaintance with numbers and skill in calculation, including the exact appreciation of time. Their habits of courtship and marriage are described; and numerous illustrations are given of the satisfaction of the maternal instinct by foster parentage.

Vol. II. is devoted to morbid phenomena, regarded by the author as of chief importance. As before, the pathology of animals is arrived at through that of man. The use of a knowledge of the subject, besides its scientific interest, is said to be for its great practical benefit, for the author believes that the efficiency of animals as servants of man depends very largely on their mental soundness, especially the training and moral care they receive; he also holds that bodily states depend on mental condition in other ways that will scarcely be believed by many, e.g., that contagious rabies is producible in the dog by exciting passions! and that the milk, blood and flesh of animals may be rendered poisonous by mere protracted suffering.

A likeness is drawn between the symptoms of animal and human insanity, the general course and a number of leading appearances being shown to be common to both, e.g., idiocy, imbecility, acute and chronic mania, with monomania, acute primary, secondary and senile dementia, various morbid impulses, such as kleptomania, erotomania, dipsomania (for many animals acquire a partiality for alcoholic excess), and perversion of the natural affections being often well marked. No mention is made, however, of epilepsy or general paralysis. But in this account of mental arrangement, mere temporary mental disturbance is too much mixed up with genuine insanity. In the matter of sleep and dreams, there seems much evidence of exact similarity between animals and man. Suicide is declared to be as common and as various in its mode of performance as in man! Several chapters are devoted to ætiology, the attempt being made to show that the causes closely correspond in animals and man; they are divided into physical (including physiological and pathological) processes, moral, and mixed.

The final section of the book is occupied with practical conclusions, consisting of directions for the training and care of domestic animals; but we cannot see that many of these rules are deserving of such deliberate and explicit statement in this book; or even that their formal laying down will lead to much practical benefit.

The means for treating animal insanity are also described; but without the mention of anything suggestive or important.

The book concludes with an appendix of a full bibliography, an interminable list of animals referred to, and a long analytical index.

F. L. Benham, M.B.

Des Paralysies dans les Maladies Aiguës. By Dr. L. Landouzy. Paris: Baillière and Co. 8vo., pp. 362.

Under the name of paralysis in acute disease has been collected a vast mass of more or less heterogeneous material, which the author of the present volume has endeavoured to sift and classify as much as possible. Though much remains to be done yet before we can hope to understand thoroughly the multifarious phenomena of febrile akinesiæ, we welcome Dr. Landouzy's work as one likely to hasten that time, were it only by clearing the ground of much ambiguity and many premature theories.

The group of paralyses under consideration is purely a clinical one, and every attempt to reduce them to an artificial unity by calling them paralyses of "convalescence," or of "asthenia," or reducing them to the "diffuse," "peripheric," "amyotrophic," "essential," "reflex," or other types, must necessarily fail. The present volume opens with some general and historical considerations of much interest; but we will at once plunge *in medias res* and follow the author in the clinical description of the paralyses occurring in various acute diseases, and then summarize with him our present notions with reference to their pathogeny and pathology.

Paralytic symptoms after diphtheria are very common, and in no way depend upon the severity or locality of the primary disease. Paterson ('Medical Times and Gazette,' 1866, p. 608) describes an interesting case, in which diphtheritic inoculation at the wrist was followed by paralysis of the four extremities. Curiously enough, the palate muscles escaped. Diphtheria is more frequently followed by paralysis in adults than in children, but the presence of albuminuria does not seem to have any causal relation to the evolution of this symptom.

The first muscles to lose power are usually those of the palate. If the nervous disturbance spreads, the eyes are affected next, then the legs and arms. When the paralysis remains localized, it usually runs a short and favourable course; but if it becomes generalized, recovery is much more protracted, though even then it may disappear in a few weeks. From every point of view, diphtheritic paralysis is characterised by a great mobility of symptoms, though Trousseau has certainly exaggerated this fact. The symptoms more particularly characteristic of a paretical condition of the palate are a nasal twang, difficulty of deglutition, of suction, of blowing, of gargling. The pharynx and larynx may become involved along with the palate. The disturbances referable to vision are due to diminution of the power of accommodation; paralysis of the muscles of the globe are much less frequent.

The legs are more commonly affected than the arms. Hemiplegia has never been shown to follow diphtheria. There are frequently troubles in the sensory sphere, paræsthesiæ, and anæsthesiæ, which may even exist without loss of motor power. Sometimes there is ataxia.

The diaphragm and other respiratory muscles are occasionally involved, as Duchenne so well showed. Dr. Landouzy gives a very interesting case in which this distinguished physician apparently saved the patient's life on several occasions by faradisation; and other cases illustrating the implication of the pneumogastric and consequent cardiac syncope.

Many French writers follow Gubler in describing a non-diphtheritic anginal paralysis. But our author thinks it more probable that the confessedly very rare cases where such a paralysis has been observed were really diphtheritic. The distinction between the diphtheritic and "herpetic" anginæ is sometimes very difficult to uphold, and the course of the paralytical symptoms in both instances is precisely the same.

The paralyses depending on typhoid fever are so various that it is not easy to summarise their symptomatology and pathology. Dr. Landouzy illustrates them copiously with cases which we can only recommend to the attention of our readers. In the majority paralysis occurs during convalescence, and appears as paraplegia or hemiplegia (with aphasia some-

times, especially in children), and is only temporary. The bladder is very often affected. Besides these disturbances, of central origin, typhoid fever may also cause neuritis and myositis, with muscular atrophy.

Dr. Landouzy discusses the pathology of dysenteric paralysis, and thinks that, in the absence of direct proof of an ascending neuritis, the clinical evidence points rather to a primarily myelitic process. On the other hand, the local paralyses (and anæsthesiæ), such as that of the orbicularis palpebrarum observed in the early stage of cholera, would seem to be rather of peripheric origin.

With reference to intermittent fever, two types of paralyses must be distinguished. The first is characterised by a transitory loss of power, intimately bound up with the febrile attack, and by aphasia, with preservation of consciousness. The second is the "hemiplegic" form of the pernicious fever of authors. It is distinguished by the severer nature of the symptoms, which are less readily modified by quinine, and persist after the febrile attack. These paralyses are probably of cortical origin, and have been explained by a mere congestive process; but it is possible that melanæmia is not without influence on their production.

In connection with variola, we occasionally find a paraplegia, or rather a paraparesis, and a number of akinesiæ, all of temporary duration, occurring during the early stage. During convalescence a variety of nervous symptoms may also supervene, both motor and sensory, diffuse or localised, sometimes with muscular atrophy or ataxia. The latter symptom has been lately studied by Kahler and Pick ('Prager Vierteljahrschrift,' 1879). Joffroy found, in a case of post-variolic atrophy of the left arm, the cord apparently healthy, though both nerve and muscles showed signs of advanced degenerative atrophy ('Arch. de Physiologie,' 1879).

Transitory paraplegia has rarely been observed as a sequel of measles. In scarlatina, early paralysis is said to be of grave omen. A case is reported, however ('Med. Times,' 1868), where a child recovered from a generalised paralysis, which supervened in the middle of the disease. The patient, it must be added, was of neurotic disposition: an illustration

of the general proposition that fevers often seize upon the *locus minimæ resistentiæ* of the organism, whence the variety observed in their manifestations. Dr. Landouzy thinks it not unlikely that, apart from actual meningeal growths, there may exist, in acute tuberculosis, a neuritis which would explain the neuralgic symptoms met with in the disease. Sciatica is not uncommon, and Eisenlohr showed it to depend, in a case lately published, on a neuritis of the sciatics.

In acute rheumatism, if we eliminate apparent paralyses and those due to embolism, paralysis due to a direct influence on the cord or nerves is rare, and, when present, fugitive and mobile. It may appear early in the disease or during convalescence.

With reference to the hemiplegias observed in pneumonia, Dr. Landouzy adopts Lépine's view, who explains them by some circulatory disturbance of the brain. They most frequently occur in old people with atheromatous arteries.

The author discusses the question of "reflex" paralyses in relation to the hemiplegias observed in the later stages of pleurisy. Under this rather unfortunate name—reflex applies to *action*, not to the *loss* of action—we include none but the cases where either an autopsy or a very rapid recovery has shown the absence of any serious spinal lesion. In pleurisy some cases of hemiplegia are clearly due to embolism, whilst others are "reflex:" that is, depend upon a centripetal irritation, producing, according to some, an ischæmia, or, according to others, a functional disturbance of the motor cells.

Until 1862, when Charcot and Vulpian published the first observation of the neuritic origin of a paralysis of the palate consecutive to diphtheria, all such akinesiæ were considered as functional or asthenic. From that day records of organic lesions in similar cases became frequent, and in 1878 Déjerine published his conclusions (see 'BRAIN,' Vol. I. p. 226), based on five personal observations. Dr. Landouzy thinks that the conclusions to be deduced from the facts actually before us are that there is a primary interference with the nutrition of the spinal cord, more particularly of its large cells; and, secondary to this, neuritis of the anterior roots,

motor nerves, with consequent paralysis. He considers the time which elapses between the febrile period and that of the paralytic symptoms as one of incubation, so to speak, during which the necessary impregnation of the cord by the poison gradually takes place.

With reference to small-pox, the observations of Westphal show the occurrence of patches of disseminated myelitis. Vulpian also describes a case of variola where atrophy of the deltoid, due probably to a localised poliomyelitis, occurred. But in opposition to the numerous cases where the clinical phenomena of a post-febrile paralysis are such as indicate a lesion of the poliomyelitic or ascending type, there are some observations on record where the most careful post-mortem examination has been completely negative with reference to any spinal lesion, whilst the peripheral nerves were the seat of distinct neuritic changes. Are we to argue from this the direct nature of the peripheral lesion? or does not analogy compel us rather to assume the previous occurrence of primary central lesions to which the neuritic processes were secondary? The slight nature of these lesions, whilst sufficient to explain interference with the trophic functions and consequent peripheral changes, would explain also their early disappearance. Indeed, why should there not be "functional" affections of the trophic, as well as of the motor and sensory, centres?

The author, alluding to the possible action of febrile temperatures in setting up molecular changes in the central grey matter, shows, on the authority of Dr. Renaut, that its hortensia, or pinkish discoloration, so often found after acute disorders, is due to an actual staining by the dissolved hæmoglobin. Might not this substance play the part of a poison, interfering with the nutrition of cells, and giving rise to the various nervous disturbances observed in such cases? It must not be forgotten either that Meynert and other observers have recorded structural changes in the grey matter, resembling in some particulars the alteration produced in myelin by overheating.

With reference to the direct influence of the specific poisons of the various fevers on the nervous centres, Dr. Landouzy thinks that the rachialgia, so characteristic in some cases,

might very well be taken as a proof of its reality. It seems to manifest itself both by immediate "functional" disturbances and secondary "irritative" lesions of the nervous elements. Future researches will have to show the nature of such poisons—in diphtheria, for instance, where figured particles are so constantly found in the blood. The variety in the localizations of such paralyses depends probably less on the acute disease itself than on certain individual predisposing causes. The asthenia of convalescence plays but a very insignificant part in the etiology of post-febrile akinesiæ, the extent of which is frequently quite out of proportion to the severity of the constitutional disturbance. The author discusses the question whether an ascending neuritis can account for the palatal paralysis after diphtheria, and shows that such a theory does not account for the cases where the disease was localized in other parts of the body, such as in wounds of the limbs or trunk, and where, nevertheless, the paralysis began among the muscles of the palate. The whole theory of ascending neuritis (or rather peri-neuritis, as Hayem has shown) is still very doubtful, and Vulpian inclines to think that if peripheral ever set up central changes, it is more probably through a functional disturbance kept up in the cord. The general subordination of the nerve-fibre to its cell, and the fact that section of a nerve, for instance, does not set up descending neuritis, but a peripherical alteration of its fibres, compel us to look on post-febrile neuritis as secondary to central changes rather than direct.

There is an important group of symptoms observed in many fatal cases of diphtheria, and referable to an extension of the paralysis to the branches of the pneumogastric supplying the respiratory and circulatory organs. Professor Revilliod, of Geneva, has expressed himself to the effect that frequently the cause of death after tracheotomy is simply a nervous disturbance. Vaso-motor troubles, changes in the rhythm of the heart, precordial anxiety, syncope, etc., which are frequently observed in acute diseases, may also be due to a morbid condition of the medulla.

From what precedes, the principles which must guide

us in framing our diagnosis and prognosis of paralyses in acute diseases can readily be gathered. Considerable difficulties may occasionally be experienced in individual cases, as similar symptoms may depend upon lesions of different nature, locality, and depth. Muscular atrophy, for instance, may be due to a myositic, neuritic, or myelitic process, the diagnosis of which may be at best doubtful; whilst the prognosis necessarily depends upon an exact knowledge of that process. Duchenne, by the way, is wrong when he describes diphtheritic paralysis as yielding normal electro-muscular reactions. The amyotrophies of febrile disease yield the reactions of degeneration, like all others; but this fact is of little use in distinguishing neuritic from spinal lesions.

In reference to the treatment of febrile akinesiæ, we must remember that in most cases they have a natural tendency towards recovery. The special indications in each case may, however, be fulfilled by acting on general principles.

Before taking leave of Dr. Landouzy's book we must mention a very full bibliographical index, which notably enhances its value. May we express the wish, in conclusion, that in his next edition the author will revise his proofs a little more carefully and remove numerous misspellings? In several places the construction of the sentences also will bear improvement. At page 275 the sense of the second sentence is far from being at once apparent.

A. DE WATTEVILLE.

General Paralysis of the Insane. By WM. JULIUS MICKLE, M.D., M.R.C.P. Lewis, London, 1880.

IT is not often that we meet with a more painstaking author than the one to whom we owe this carefully compiled monograph, or one who more conscientiously records the theories, hypotheses, and the guesses at truth of each and every one who has mingled in the maze of observation, investigation, or interpretation of this most interesting and perplexing disease. If it was not much to the credit of the medical profession that

so remarkable and well-marked a disease as general paralysis should through all the centuries have remained unrecognized until, in the words of our author, "the discovery burst forth with full effulgence in the works of Bayle," in 1822. We fear that the full effulgence has since that time been not a little obscured by rough and rude observation and by rash hypothesis. Every one almost engaged in the custody of the insane appears to have thought that he was able and entitled to make incursions of discovery in this *terra incognita* of pathology, even if he only brought back one pebble to cast upon the cairn of ignorance, which Dr. Mickle has taken so much trouble to count, measure, and describe, that he has scarcely attempted the higher and far more essential task of appraisement and discrimination. Up to the middle of the volume the foot-notes of reference constitute quite a bibliography of authors, many of them unknown, or known only to be mistrusted, and the same profusion of references is continued throughout to such an extent that to anyone not conversant with the personality of its literature, this preamble of opposing facts and discordant opinions greatly detracts from the real merit of the work, which consists in the careful record of the rich experience of its author, and in his generally sound and judicious inferences. Personally we respect Dr. Mickle all the more for this fault of his book, just as we are compelled personally to disesteem the psychological author of the opposite type, who, utterly devoid of all originality, succeeds in writing readable books which are a tissue of the threads of thought stolen from other men. But the pleasant thief may even be more profitable reading than the conscientious compiler, because his art enables us to grasp the ideas which he has assimilated, and thus we may feed upon Milton or upon any other of the monarchs of thought, dished up in a modern *menu*, and we may reflect, as Hamlet did, how that a man may "eat of the fish that hath fed of that worm, that hath eat of a king," to show that "a king may go a progress through the guts of a beggar."

But the fault we find with Dr. Mickle is that he contradicts his name, and makes not enough of himself, by sandwiching his opinions between the coarse bread and the bad butter or the oleomargarine of others. It is with difficulty that we get

a taste of them, yet when we do, the flavour is right, for he is evidently a careful observer and honest thinker, and he has grand opportunities of observing at least the later stages of the disease about which he writes; for Dr. Mickle is the medical superintendent of that proprietary lunatic asylum at Bow which belongs to a private gentleman named Byas, and in which, under the sanction of the Commissioners in Lunacy, the Government causes its soldiers who become insane to be incarcerated. The history of the treatment of insane soldiers deserves to be told. For a time the army authorities borrowed the hospital at Yarmouth from the navy, but when it was wanted for insane sailors, and when, moreover, it had been discovered that soldiers becoming insane could not be got rid of by turning them loose in the streets of Chatham, after the parish authorities had been advised that they must take care to apprehend these loose madmen as not being under proper care and control—then the insane soldiers were farmed out to Mr. Byas, who has had the good sense to employ Dr. Mickle to take care of them. The army has hitherto been supposed to contain its full proportion of intemperate and dissolute men, and as drink and dissipation are supposed to be efficient causes in the production of general paralysis, Dr. Mickle has enjoyed a wide field of observation on the subject of his monograph, of which he has not failed to avail himself.

The first chapter, on what the author calls the Prodromes, valuable as it may be, does not make up for the defect we feel in the more important chapter on Diagnosis that the latter is instituted in regard to the fully developed form of the disease, where small difficulty is usually met with, and little skill required. There are few, if any, of the physical prodromes which may not prove to be precursory signs of other disorders, or which may not pass away without the supervention of any diseases. The most important and significant precursor is "sudden unforeseen moral falls, of which theft is one of the most frequent, which occur to those hitherto without reproach. In the history of many a case do we find that some moral or other mental change in the patient has been noticed long before the recognized onset of the disease." We may observe in passing that these so-called moral falls appear to indicate

general change of the mental faculties, and that this kind of thief does not steal because his desire has overcome his sense of right, but simply because he has misjudged his relation to the property of others. The so-called moral perversion is in truth insidious dementia, notwithstanding the fact that no little ingenuity and design are sometimes displayed in obtaining the proposed end. But in judging of the medical purport of "sudden unforeseen moral falls" without the accompaniment of physical symptoms, the mental physician has great need to beware lest he be used as a cat's-paw on behalf of some real criminal who may for long have broken many or most of the Commandments.

In the important chapter on symptoms, as distinct from precursors, we are dreadfully puzzled to know just where we are, in consequence of the author's habit of running a little way after everyone's lead, and then forsaking it for some new track. The one important thing to do was to fix and define the very earliest symptoms by which this fatal disease can be definitely recognized; but, instead of doing this, the author discusses the stages and periods of the disease according to the diverse opinions of several authors, in whose observations and opinions we should have much less confidence than in his own, if he would only have given them without all this frippery of reference. It is a mistake to describe a continuous disease like General Paralysis as if it had stages like a coach, or even like an exanthematous fever; and it is an abuse of language to discuss periods in a progress where there is but one period—that is, death. That which would be most precious in practice, but which we do not get, would be an original investigation into the earliest trustworthy signs and symptoms, and into the just value of those which are more or less reliable. In default of such guidance, mistakes are constantly being made even by practitioners of some experience, while by the profession at large the early recognition of this disease is a task as hopeless as it is important. In default of such knowledge, it can be no matter of surprise that the chapter on Diagnosis points to the roughest discrimination from the most unlike diseases. It may be worth while to tell a man that if he walks upon his own shadow he will not go to the south

in these latitudes; but in medicine an exact semeiology is a mariner's compass which supersedes the need of such rough directions; and for once in a way the author does object to one of his recent authorities, Voisin, for including senile dementia in General Paralysis, and to the "regrettable tendency in this direction, with the result of thrusting into the domain of general paralysis a host of various senile cases alien to it."

In his account of the microscopical appearances, as described by Mierzejewski and others, the author says:—

"So great is the variety of morbid appearances described, and of the localities said to be principally affected, that I propose to trace the changes throughout the whole central nervous system, beginning at the brain as a matter of convenience, and without prejudice as to what part of the nervous system is the primary or principal seat of the disease—a question as to which the most conflicting replies have been returned by workers in nervous pathology. With facility one might dogmatize; but in the present imperfect and shifting state of our knowledge, I think it better to quote several observations, even at the risk of being thought tedious."

Therefore with regard to the Pathology and the Pathological Physiology, we are duly informed of the views entertained by Lubinoff, Luys, Lionville, Lasèque, Linas, Landors, Lusana, Lemoine, Landouzy, and other writers of as great authority; the result being that "our attention is claimed by a conflict of opinion as to whether the essential pathological change in general paralysis is principally and primarily of a more or less frank inflammatory nature, or principally and primarily of a degenerative nature." And it is no wonder that the author concludes that, "confronted with these various and often irreconcilable views, we may proceed to examine the subject anew." The wonder rather is that he should have imitated the frequent habit of many insane patients by such promiscuous accumulation.

Forced in this manner to examine the subject anew, the author proposes a view of his own, which is that "the morbid process in the nervous system first deranges and then destroys, or tends to destroy, the functions of the parts affected.

"In the vast majority of cases the cerebral cortex is

primarily affected, the meninges usually being more or less involved almost simultaneously; in many cases the morbid process is most active in circumscribed regions; the convolutions of the frontal and parietal lobes suffer more than other parts." There is repeated and more or less persistent cerebro-meningeal hyperæmia; distention of the vessels, circulatory impediments, irritative over-growth of the connective nuclei of the walls of the vessels, and probably also of the neuroglia; while others of the nuclei and cellules, often termed embryoplastic, or their materials, are perhaps directly effused. Other changes from the lowered standard of the local processes of nutrition are wandering of white blood-corpuscles, and escape or extravasation of red blood-corpuscles; tendency to diffuseness of all the morbid processes, and thickening opacity and œdema of the superjacent meninges; the nerve-cells failing in their nutrition, and hence their swelling cloudiness and final degeneration. Then, if a chronic, mild, adhesive form of inflammation sets in, fibrinous effusions and production of connective tissue assist in more completely involving the nerve-cells and in tying down the membranes to the cortex. "These changes proceed in the usual degenerative course, and finally, as a result of them, we find the processes of the nerve-cells cut off by the way—the cells themselves atrophic and degenerate—the blood-vessels fatty, calcareous, pigmented, and misshapen, and the former hyperplastic neuroglia now atrophied."

Such is the author's "own view," somewhat abbreviated, of the pathological changes; and this "own view," as it bears upon the course of the disease, is continued for many pages well worth reading and reflecting upon. We can only regret that the volume does not contain a much larger proportion of statements, suggestions, and opinions from the same source. The chapter on Therapeutics, which comes to us nearly undiluted by references, is very sound and sensible. The author thinks highly of veratrum viride during the earlier periods, and of perchloride of iron throughout; and he thinks prolonged artificial suppuration likely to do good, but has not had the courage to try. We have frequently used it, and have been convinced the progress of the disease was

retarded thereby; but, like Dr. Mickle, we have never seen any treatment followed by any better result than a temporary remission of symptoms. Still there can be no doubt that appropriate treatment will greatly diminish the apparent sufferings of the patient, or at least render them less inconvenient and distressing to those about him.

The volume is concluded by a small-type Appendix of cases, in which the author attempts to establish five varieties of the disease, and it forms a valuable record of well-observed facts. It seems to be pretty certain that there are no kinds of general paralysis distinct from each other. Every case is dissimilar, within certain limits, from all other cases, but it may be doubted whether each group does not pass by insensible gradations into other groups; and the author fully recognizes the principle that the essential pathological process is not multiform; and if he only makes use of his attempt to group the individual cases as a means of investigating the causes of their difference, the labour may be well repaid. During further inquiry he will probably have to rearrange his groups many times, in order to make them fit in with the varying facts he may meet with. The groups Nos. 3 and 4 seem to be mainly based upon the encephalic localities of the pathological changes. No. 2, upon the chronicity and mildness of the disease. No. 5, upon a local cortical induration. No. 1, upon generalized and symmetrical affections of the hemispheres, with adhesion and decortication of the cerebellum, but the plan is too complex to promise success as a method of investigation. The groups should have been formed on some consistent plan: for instance, the localities affected, or the kinds of morbid process. Grouping in a methodical manner is by no means an inefficient means of investigation, but grouping according to any characteristics of the most diverse kind which seemingly present themselves will be likely to disappoint the hopes and efforts of the observer.

JOHN CHARLES BUCKNILL.

Clinical Cases.

CASE OF MICROCEPHALIC IDIOCY.

BY A. R. URQUHART, M.D.,

Medical-Superintendent, Murray's Royal Asylum, Perth.

IN addition to the two cases of Microcephalic Idiocy described by Dr. H. R. O. Sankey in this Journal ('BRAIN,' October 1878), there is at present a third patient similarly affected under the care of Dr. Parsey (Warwick County Asylum).

E. F. was admitted into the Warwick County Asylum on the 20th of September, 1876, from a remote workhouse, where she had been detained for five years.

Mr. Hitchens, of Shipston-on-Stour, has kindly obtained the following facts from her father. Her parents were not related before marriage, and were of average intelligence, sober and industrious. Her father is now seventy-four years of age, a hale old field-worker. Her mother died of phthisis twenty-five years ago, was always delicate, and for the last fifteen years of her life did but little work.

She had seven children, three—still alive and without mental defect—born before E. F. Two born after E. F. were also healthy, but one of them died, æt. 16, of rheumatic fever. The last child was seven years younger than the patient, and had an abnormally small head, though not so small as hers. This girl had more intellectual capacity than E. F., but she never walked, and suffered from general marasmus till her death, when nine years old.

The family, as a rule, appear to be fairly healthy. The only ascertained history of nervous disease occurred in a niece of the mother's, who had a paralytic seizure.

During Mrs. F.'s pregnancy with E. F. she had no disease, and met with no accident; and there is no history of any extraordinary maternal impression. At birth the cranium of E. F. was noticed to be very small, the medical attendant stating that her brains were all in the back of her head.

When in the workhouse she was very passionate, noisy, and excitable; and was reported in the certificate of insanity to be getting more unmanageable.

On her admission she was in a similar condition to her present state, though during her residence here she has become quieter and more tractable, and has also improved in her habits.

She is now forty years of age, but looks considerably older. Her attitude is peculiar. She perches herself on the edge of a chair in a simian posture, the hands being invariably crossed on the breast, constantly intertwining in a nerveless, purposeless way to an accompaniment of a rhythmical swaying of the body to and fro. Her back is bent till her head appears to be set on at right angles to her body, and her neck is usually on the full stretch; she is either craning forward in her contortions or her head is bent down to the utmost. In summer her chosen place is by the corridor door, in winter hard by the fire. She is shrivelled and aged-looking. The wrinkles on her face are deep, and her breasts are withered and pendulous. She is slightly built, with small bones, and insignificant muscular development. Her habit of stooping has brought her shoulders forward to the utmost, but otherwise she presents no extraordinary deformity of body or limbs, save for a slight inequality in the length of forearm and hand—the right being the longer by half an inch. Her head at once attracts attention by its minute size, which somewhat luxuriant grey hair is perfectly inadequate to compensate for. Her complexion is sallow, and her expression in repose fatuous in the extreme. At other times it varies between fear and pleasure, rarely showing attention or curiosity.

The shape of her head is oxycephalic, tapering to a cone-like summit. Her nose is long and prominent, surmounted by well-developed frontal sinuses, immediately above which the frontal bone bends suddenly backwards, and slants upwards on a parallel plane with the nose. The remaining upper incisors project from the alveolus and the chin suddenly retreats beneath a wide and capacious mouth. The cheek-bones form the widest part of the face. The occipital spine is boldly marked, and forms an excellent tubercle for measuring from. The cranial sutures are closed.

She walks in an exceedingly clumsy manner, lifting her feet high from the ground and rolling at each step, She runs but seldom, and when she does, it is with an indescribable shuffle, with head thrust out in front and a feeling of tottering insecurity. All her motions are clumsy and express a want of co-ordinating power. Notwithstanding she possesses a fair amount of strength, and in anger has been known to pull

another patient down on the floor. Her grasp is apparently feeble under ordinary circumstances, and her intelligence so limited that she cannot exert it on command. It is easy, for instance, to pull a tea-cup out of her hands, but the nurses sometimes find it rather difficult to persuade her to enter a bathroom-door. She claps her hands sometimes, but without eliciting much noise. Though ravenous at meal times, her haste in eating is owing to the quantities she stuffs in her mouth, not to the rapidity of arm motion.

Her days are spent sitting, as above described, on a chair, from which she very rarely moves unless when summoned to meals or to bed. She is passionate when tormented, but appears on the whole to be of an amiable temper, with the characteristic confiding simplicity of idiocy.

With regard to her bodily condition, she is in fair health. On her admission she was often sick after food, but now this rarely happens. Her tongue is clean and moist, her appetite good, her bowels regular. The teeth are yellow and chalky, and several have dropped out. The palate is low and smooth. Her skin is moist and free from eruptions.

The organs of circulation present the usual characteristics of congenital idiocy. The pulse is slow, weak and small. The heart's impulse is feeble, the sounds normal. The feebleness of circulation is manifested by the constant swelling, œdema, and lividity of the feet and legs, which sometimes indeed necessitates her detention in bed.

Respiratory organs apparently normal. The inspiration is extremely shallow and feeble.

She used to menstruate at irregular intervals till about three years ago, when the catamenia suddenly ceased for six months, at the end of which time she suffered from a menorrhagic attack, after which all menstruation ceased. She has never been known to manifest any sexual feelings. Her genital organs are apparently normal. Her sight is intact, for long and short range of vision. Hearing is also apparently perfect. Smell is but feebly developed. Taste is evidently present—she prefers bread and treacle to bread and butter—and touch is sensitive if not indeed hyperæsthetic.

Her intelligence is extremely low. She recognises strangers, if in a different garb to the ordinary asylum dress; and she knows, at any rate, one patient who is her especial aversion. She recognises her name, also simple words and phrases—as, dinner, bed, bath, music, &c.—but her vocabulary is extremely limited.

She is perfectly helpless in regard to dressing herself. The only use she can make of her hands is in feeding; she can use a spoon.

Her language is confined to interjections; her whole stock being, "Oh dear!" "Now then!" "Ah = Yes!" "Hulloa!" "Ah!" and "No." And these are pronounced so indistinctly that it requires some experience to determine which is which. When asked to go to the bath, she will sometimes say "No." She is fond of pretending that she is ill, saying, "Ah!" with indrawn breath and a very good imitation of suffering expressed by hands pressed on stomach and piteous noise.

Attention is hardly present. It requires considerable motor power to rouse her attention, and then it is but a momentary exhibition. For instance, she can be roused to scream at the sight of a penknife being opened, but would take no notice of a dog in unaccustomed attitudes.

There does not appear to be any imitative faculty in force.

She has no fund of amusements, no fondness for any toy or ornament. A doll will lie in her lap unheeded, and picture-books are beyond her range entirely.

She seems to have a preference for little children, and has on more than one occasion given them portions of bread and butter.

Her memory is certainly rudimentary. She remembers having an abscess in her leg opened by a lancet two years ago—witness her screams on seeing a penknife; she certainly recognises tape and callipers, and has a lively recollection of my stealing her bread and butter at tea.

Her fondness for music is remarkable. This is evidently not a mere liking for noises; but she can really appreciate musical rhythm. Her great delight is to hear the band play, and when the music is in full swing her contortions are most pronounced, and her grunty noises are at their loudest.

Identity is present—evidenced by her knowledge of her own ward, her own bed, and her own seat.

She would pay no attention to the calls of nature; and is only preserved clean by being taken to the closet daily. She never makes any attempt to find her way there herself, but when there gives no farther trouble.

She is very timid and easily frightened.

Exhibits no affection or preference for any single patient. In fact her habits are perfectly solitary.

She still continues very passionate, though her opportunities of showing this have been much diminished. She is also revengeful as regards one fellow-patient only. This girl has a most objectionable habit of staring into her neighbours' faces from a very close proximity, and E. F. will at once take the offensive on her approach. With an evident memory of former encounters, she makes hideous noises

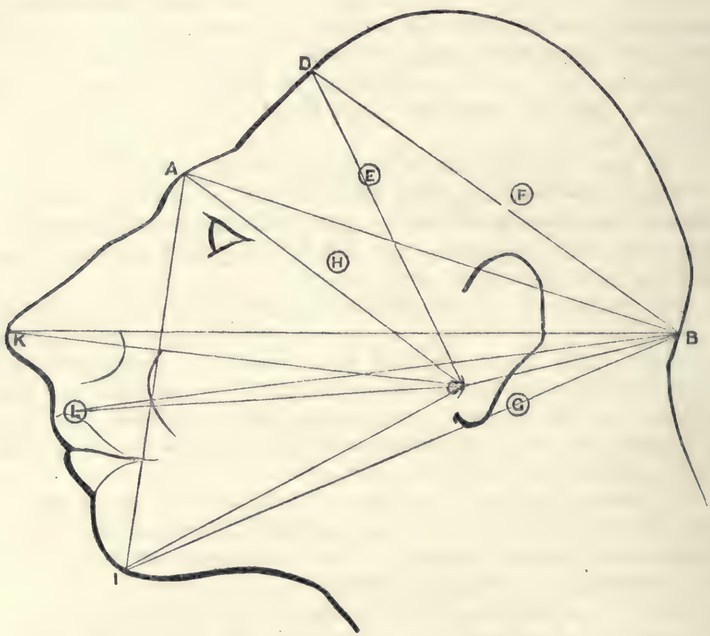
and clutches at the object of her wrath with a ferocity that could scarcely be credited to her slight figure and nerveless fingers.

Occasionally, though now not so frequently as in former years, she lies down, refuses to move, and has to be carried to bed.

Curiosity is very faintly developed.

Pleasure is manifested by smiles and a quickened rocking of the body, and a more rapid interlacing of fingers. This is chiefly manifested on hearing music.

She often exhibits mortification and jealousy when she is overlooked by the nurse or her fellow-patients going out to the garden. At these times she becomes noisy, and is only pacified on being allowed to join her neighbours.



DIAGRAM—HALF LIFE SIZE—OF HEAD MEASUREMENTS.

Sympathy, and all the more complex psychical phenomena are quite beyond her ken. In fact her mental capacity might be rudely gauged at something below that of a fairly intelligent house-dog.

She sleeps in the fœtal position, covered entirely by the bedclothes.

MEASUREMENTS.

Height			4 feet 9½ inches.	
Weight			85 lb.	
Head.	Curves	Circumference (A B)	16½	inches.
		Antero-posterior (A B)	9	„
		Bi-auricular (C C)	10	„
	Forehead	Breadth (E E)	3⅞	„
		Height (A D)	1¾	„
	Diameter	Longitudinal (A B)	5¾	„
		Transverse (F F)	4¼	„
		Bimastoid (G G)	4	„
		Bizygomatic (H H)	4⅛	„
		Fronto-mental (A I)	4	„
		Occipito-frontal (B D)	5⅛	„
		Occipito-superciliar (B A)	5¾	„
		Occipito-nasal (B K)	7¼	„
		Occipito-alveolar (B L)	6⅜	„
	Callipers	Tragus to chin (C I)	4⅞	„
		„ Alveolus (C L)	4⅛	„
		„ Nasal prominence (C K)	5	„
		„ Superciliary ridge (C A)	4⅛	„
		„ Top of forehead (C D)	4¼	„
		„ Occiput (C B)	3¼	„
	Curves	„ Chin (C I)	5¼	„
		„ Alveolus (C L)	4¾	„
		„ Tip of nose (C K)	5¼	„
		„ Superciliary ridge (C A)	4¾	„
		„ Top of forehead (C D)	4⅝	„
		„ Occiput (C B)	4½	„
		Palate width	1¼	inches.
	Ears	Length	2⅝	„
		Breadth	1⅜	„
		Length of Arm	11	„
		„ Forearm	9	„
		„ Hand	6½	„
		„ Thigh	16¼	„
		„ Leg	17½	„
		„ Foot	8½	„
		Round Arm	7½	„
		„ Calf	10½	„
		Width of Shoulders	10⅛	„
		„ Pelvis	10½	„

CASE OF CEREBRAL TUMOUR.

BY ARTHUR W. FOX, M.B.

Physician to the Eastern Dispensary, and Bellott's Mineral-Water Hospital, Bath,

and

ERNEST FIELD, M.B.

Physician to the Eastern Dispensary, Bath.

H., a shoemaker, aged 42 years, consulted me on September 14th, 1878. He was of middle height, muscular build, pale complexion, and had a stolid expression of countenance.

He stated that nine years ago, when he was fighting with another man, he tumbled down and was unconscious, but that he was not knocked down. His wife informed me that the true version of the story was, that he had been playing at cards with some other men, when a quarrel arose, which ended in his being knocked down and kicked violently in the head, and that he was so stunned as to be unconscious for a long time. Two years and a half ago, when at breakfast, his "face turned very pale, and his lips black," but he was not unconscious, neither had he headache, or twitching of his limbs, but he felt giddy. Since then he has often had these "pale fits," sometimes as many as two or three attacks in a day. On one occasion a fortnight elapsed without his having any. He was obliged to give up work a fortnight before Christmas, 1877, on account of violent pain in his head in the frontal region; for this he became an out-patient at the United Hospital, when, as he required to be visited at his own home, he was under the care of Dr. Field, who found, upon examination of his optic discs, double optic neuritis; and, coupling this fact with the violent headache, he formed the opinion that the case was one of cerebral tumour.

The patient was able to read up to the date of a month before Christmas, 1877; since then he has gradually become blind. There was no history whatever of syphilis, but he had gonorrhœa many years ago. He drank to excess when he was

younger, but has not done so for years. His mother died of phthisis, and he has a cousin insane.

Present State.—Mr. Mason, who recently examined his eyes at the eye infirmary, describes his discs as in a state of white atrophy. His pupils are contracted, equal. Smell unmistakably blunted on both sides. Taste fair. Hearing good. Memory affected as to recent events, but good for what happened some time ago. Sleeps well. Complains of constant headache in frontal, and to some extent parietal region, but he is no longer liable to the violent paroxysmal exacerbations he suffered from when he was under Dr. Field's notice. Tongue protruded straight, and moved well in all directions; organ in a state of fibrillary tremor. No appreciable tremor of palate. No affection of speech. No nystagmus. Fine tremor of both arms, which grows coarser upon an attempt at voluntary movement. Grasp of hands equal. No paralysis of face, limbs, or sphincters. This is neither anæsthesia nor hyperæsthesia. The reflex excitability of skin and tendons is normal. Although blind, he is able to touch with precision various parts of his face and body. Appetite good; never any nausea. Bowels regular. Urine, specific gravity, 1027; trace of sugar. Other systems apparently normal.

October 2nd. Had a severe general convulsion. When I saw him he had regained consciousness; he complained of severe pain in his head in the frontal region. His face and head were dripping with perspiration.

November 3rd. According to his wife's statement, he had an attack without convulsion, in which he was unconscious; when I saw him, shortly afterwards, he was quite conscious. In addition to the usual tremor of his arms, there was general tremor of head and body; no nystagmus.

November 11th. Another severe fit, in which he was unconscious. His wife described it as follows: First, rigidity of both legs, but the right leg the most rigid; left arm flexed and rigid; head bent forward: this was followed by shaking all over. Unfortunately there is no note of the period when he became unconscious. His breathing was loudly stertorous. When I saw him, half an hour afterwards, he had regained consciousness. There was no paralysis, but there was a coarse tremor of arms and legs; none of head, and no nystagmus. Nothing could convince him that he was in his own house.

December 2nd. Vomited. In the evening had a severe convulsive seizure, and was a long time unconscious. At the commencement there was rigidity of both legs, and both arms were flexed.

January 21st. Has lately had what his wife terms "sleepy fits," and several of his former "white fits." Her description of

a general convulsive attack he had to-day is as follows: "His face first reddened, and he then became unconscious. His eyes protruded, and his face became pale. His eyes then rolled, and, at the same time, his limbs became rigid. This was followed by shaking all over." His wife also told me that when he is in his sleepy fits, in which he is only partially unconscious, his "head is drawn backwards when the fits begin," and at the same time he has a "twitching of his left shoulder." She describes it as an upward movement. When I saw him some time after his severe attack, he told me that he had no headache; he appeared much as usual. His wife states that when he walks he tends to fall backwards. He had some difficulty to-day in passing water.

April 11*th*. Has lately had several "sleepy fits" and "white fits," but has had no seizure in which he has been completely unconscious. The tremor of his arms and the fibrillary tremor of his tongue remain about the same as when I first saw him. On this occasion the tremor of right arms is coarser than that of left. Grasp of right hand stronger than that of left. Recently, when he has wished to pass water, he has occasionally been unable to do so, but when the stream comes he is unable to control it.

From this date I never saw him again, as he went into the union under the care of my friend Mr. Biggs. I learnt subsequently, that during his residence in the union, he complained but seldom, if ever, of headache, and he became very fat. He had no paralysis. He was happy, but had a delusion that he had been at home "mending and making boots," and "that he was put in the wrong bed,"—an old delusion of his. He never had any delusions of grandeur, or apparently any feeling of exaltation. He died in a general convulsive seizure on March 25th, 1880, at 6.30 A.M.

The post-mortem was made fifty-three hours after death, on March 29th, by Dr. Field, in the presence of Mr. Biggs and myself. The following account is taken from Dr. Field's notes: —Scalp very thick, much congestion of vessels. Cranium moderately thick and hard, contents tightly packed; some flattening of convolutions, vessels of membranes full. Whole brain rather firmer than normal, but anterior right half of hemisphere very hard. On making horizontal sections through the right hemisphere, a cribriform appearance was seen in the white matter covering the corpus striatum, and this was still more marked in the septum separating the anterior part of the two lateral ventricles, where cystoid spaces existed in numbers, some the size of a threepenny piece, and one that of a sixpence; these spaces contained a colourless fluid. The cribriform tissue was much thickened, and much firmer than the rest of the brain.

The right corpus striatum was very hard, and larger than the left, but in colour was normal, as if produced by an increase of the connective tissue. Part of the orbital convolution on the right side, bordering upon the Sylvian fissure, was pushed out into a protuberance, resembling an epithelioma in shape, and was composed of the same firm tissue as the other hardened parts. The hardening excavating process had extended slightly to the tissue covering the left corpus striatum, but it was only slightly marked as compared with the right side. All other parts of the brain, the cerebellum, and the vessels, were normal. The optic nerves were normal in appearance, not hardened. The growth in the right corpus striatum consisted of numerous small granules disposed in a stroma of fine reticulated connective tissue: in the unstained sections the structure could with difficulty be made out; but logwood at once differentiated the elements, and rendered the nature of the growth evident. The general appearance of the tissue was quite that of a hyperplasia of the neuroglia normally existing between the nerve elements, and differed considerably from that presented by a round celled sarcoma, with which glioma is frequently considered to be identical: the small cells of which the growth was mainly composed exactly resembling the granules of the neuroglia, and being much less in size than the majority of the cells met with in sarcomatous tumours. The enlarged orbital convolution had the same structure as the right corpus striatum.

Remarks.—There are but few remarks to make upon this interesting case; and I must confess to having been in considerable doubt as to the nature and situation of the cerebral lesion.

That coarse intra-cranial disease in all probability existed was clearly shown by the history of the patient, his headache and double optic neuritis, though the presence of the latter does not invariably indicate some adventitious intra-cranial product, as has been pointed out both by Drs. Stephen Mackenzie[1] and Hughlings-Jackson.[2] Regarding the etiology of the case, there was certainly no history of syphilis, for I carefully went into the question; and a tubercular tumour appeared to me to be excluded on account of his age, the absence of pyrexia and of symptoms pointing to tuberculosis of other organs; also the long duration of the disease, and the fact of his gaining in condition. As to carcinoma it only required to be thought of to be at once rejected. On the whole I was inclined to look upon his symptoms as probably due to a glioma, or to some

[1] 'BRAIN,' July number, 1879, p. 217.

[2] The Royal Ophthalmic Hospital Report, vol. viii., pt. iii. p. 445.

sclerotic process; in favour of glioma was the long duration of the disease, and his maintaining his state of good nutrition. With regard to the localization of the disease, it was impossible, in my opinion, to come to any conclusion, as the only constant motor symptom was the fine tremor of arms, legs, and tongue, which became coarser upon an attempt at voluntary movement, and in that respect resembling the tremor of multiple sclerosis, but differing from it in the smallness of the oscillation, and, with the exception of the tongue, as a rule not extending to the head. It certainly appears to be a remarkable fact, considering the position of the growth, more especially its infiltrating the right corpus striatum, that he had no hemiplegia, and, excepting on the occasion when I last examined him (April 11th, 1879), any diminished power in the grasp of the left hand, but it only confirms Obernier's[1] statement, that the intensity of the symptoms produced by a cerebral tumour are in direct ratio with the rapidity of its growth, and vary greatly according to individual irritability; also it is impossible to compare slow growing tumours, which may press aside without destroying nerve paths, with those lesions, which in their rapid march destroy whole territories of the central nervous system. The mental symptoms exhibited by this patient were partly due, I imagine, to the changes we found in the profrontal lobes (orbital convolution); as injuries, and diseases of that region, according to Dr. Ferrier,[2] are not followed by paralysis of motion or sensation, but by psychical disturbances of various kinds. The partial bilateral anosmia, I believe, was simply the result of brain squeezing.

[1] 'Cyclopædia of Medicine,' Von Ziemssen, vol. xii. p. 241, 243, 264.

[2] Dr. Ferrier, 'Gulstonian Lectures on the Localization of Cerebral Diseases.'

(Read before the Bath Pathological and Clinical Society.)

Abstracts of British and Foreign Journals.

Method of Preserving the Brain.—Professor Carlo Giacomini of Turin has lately described a process for the preservation of the Brain and Nerves which promises to be of great utility to the anatomist and pathologist. The description of the process was communicated to the Royal Academy of Medicine of Turin on the 7th June 1879, and as I lately had an opportunity of seeing the results of the method employed, and was much impressed with its success, I think some account of it may be interesting to the readers of this journal.

After referring to the various methods previously proposed, such as, 1, the plan of Broca (1860), of hardening in nitric acid diluted with nine parts of water, and subsequent desiccation; 2, that of Frederig of Ghent, modified by Duval of Paris, of successive immersion in dilute nitric acid, chromate of potash, alcohol and liquid paraffin; and 3, the plan of l'Oré of Bordeaux, by immersion in alcohol, subjection to a temperature of 45° C. for 15 or 20 hours, varnishing with gum elastic, and finally electroplating, and stating the insufficiency of them all for the convenient preservation and subsequent examination of the brain, Professor Giacomini explains the manner in which, from a knowledge of the properties of glycerine, he was led to make use of this substance for the preservation in the moist state of brains which had been previously indurated by other reagents.

The primary induration of the brain may be effected by one or other of several reagents, such as chloride of zinc, chromate of potash, or chromic acid solutions, diluted nitric acid or alcohol, but of all these the saturated solution of chloride of zinc appears for several reasons to be the best.

The newly extracted brain is placed in a saturated solution of the chloride of zinc, with the membranes still adhering to it; and after an immersion for about 48 hours the membranes and blood-

vessels may with ease be removed, care being taken to preserve the surface as entire as possible. The brain at first naturally floats in the solution, so that it must be turned in it from time to time till the whole is acted upon, and from the increase in specific gravity it at last sinks in the solution. This generally occurs after two or three days' further immersion, and then the brain is to be transferred from the solution to alcohol of commerce. By this part of the process the brain loses about a twentieth or twenty-fifth part of its weight.

Should there be reason to believe that the brain has suffered from the body having been kept too long after death, or if it is wished to examine it *in situ*, the induration may be effected by the injection of 600 grammes of the saturated chloride of zinc solution into the internal carotid arteries.

The brain when transferred to the alcohol sinks in that fluid, and precautions must be taken, by frequently changing its position, to prevent any alteration of form by pressure against the sides or bottom of the vessel in which it is laid. After remaining ten or twelve days in the alcohol, the brain is ready to be transferred to glycerine.

For this purpose white or colourless fenicated glycerine (in the proportion of 1 to 100) is found to answer best. The brain must remain in the glycerine for a number of days till it is thoroughly impregnated with it, when it will be found to have regained from 5 to 6½ oz. in weight. It is then removed from the glycerine, and placed in any convenient situation, to allow of some desiccation and the exudation of superfluous fluid, and it may either be preserved in this state, or, what is better, it may be covered with one or more layers of a varnish of caoutchouc or marine glue, which, while it does not materially alter its appearance or consistence, protects the surface from the adhesion of dust and from mechanical injury, and enables the preparation to be handled pretty freely without risk of breaking the surface. The final varnishing is not however necessary if the preparations are otherwise fully protected.

Preparations made after the manner now described may be kept for years in close glass or other cases without change; and such is the hygroscopic property of the glycerine that they may even be left exposed for a considerable time in ordinary states of the atmosphere without undergoing any marked alteration in weight or appearance. The natural form and colour of the brain are in great measure maintained, and even the distinction of the grey and

white substance, and the fibrous character of the latter are preserved in such a manner that thin slices can be made and dissections of the preserved brains, much in the same way as in a fresh brain, or in one which has been successfully preserved in alcohol.

It is true the microscopic characters of minute structure are not perfectly preserved; but this could not have been expected from any other methods than those adopted by histologists in their improved modern processes. But for the conservation of series of human brains and those of animals for purposes of exhibition and demonstration, and for the study of the external form and larger structure, they are admirably adapted and likely to prove most valuable, for they remain to some extent soft and pliable, can be handled with freedom, and may be dissected or examined in every way that may be desired.

It is obvious that series of brains so preserved are remarkably well adapted for the study of the comparative anatomy and homology of the sulci and convolutions both in man and animals, and that they are equally well suited for serving as records of the local effects of pathological changes or of physiological experiments.

It seems unnecessary to follow Professor Giacomini further in his description of the varieties in his method of preliminary hardening by other reagents, such as chromate of potash, chromic acid, &c., as that by means of the chloride of zinc appears to be on the whole the simplest and most satisfactory.

I may mention further that in the Anatomical Museum of the University of Turin I saw also with Professor Giacomini elaborate isolated dissections of the whole and parts of the central nervous system, in connection with the brain and spinal marrow and with the nerve roots, ganglia and plexuses, and the anastomoses and subdivision of branches of nerves displayed with great beauty and accuracy, and all preserved in the moist condition after the same manner as the brain preparations, and exhibited under glass covers; and I could not but regard these as most useful and illustrative preparations both for demonstration and study, and especially as immeasurably superior to the older preparations of the same kind in the dried condition.

ALLEN THOMSON.

Krueg on the Sulci of the Zonoplacental Mammalian Brain. (*Zeitsch. für wissenschaft. Zoologie*, Bd. 23. 5 Plates.)—In

this memoir, relating chiefly to the carnivora, the cerebral sulci are divided into three groups.

1. Boundary-sulci (Grenzfurchen) (three in number) which divide the hemisphere into histologically different regions, and which are constant, not only in carnivora, but in all mammals, whether smooth-brained or not.

2. Primary sulci (nine in number), which are present in all species of carnivora without exception.

3. Secondary sulci (thirteen), which are variously arranged in different groups, and in some are accessory or wanting altogether. The individual families of the carnivora may be identified with tolerable precision by the presence and disposition of these secondary sulci.

Further, Krueg indicates homologies between the sulci of the carnivora and ungulata, and renders possible a thorough-going comparison between these large mammalian groups. The work is based on the developmental history of the cat and dog. In addition there are depicted in the plates the brains of forty-seven different species.

H. Obersteiner.

Sur le Développement du Cerveau, &c. Ch. Féré. (*Revue d'Anthropologie*, 1879, No. 3, p. 661.)—The author gives measurements of certain cranio-cerebral relations in the fœtus and young child (sixty subjects), with especial reference to the relative development of cerebral regions. By his method pegs are inserted through the fontanelles to assist in maintaining the normal relations, and the parietals are reflected outwards; the falx prevents separation of the frontal and occipital, and so maintains the ant. post. diameter of the brain, while the bulging lateral diameter can be temporarily restored by replacing the parietals. Topography is not satisfactory before the 5th fœtal month; at the 6th, the fissure of Rolando is unmistakable. Contrary to what has been affirmed this fissure is throughout *posterior* to the coronal suture, and converges to it from above downwards, as in the adult. The Sylvian fissure passes *above* the squamosal bone, and the parieto-occipital fissure lies *in front* of the lambda—while in adults the Sylvian f. very nearly follows the ant. sup. half of the squamous border, and the lambda is nearly always on a level with the parieto-occipital f. From these relations Féré inclines to think that in the infantile brain the occipito-spheno-temporal region, "the sensory and vegetative region" takes precedence in growth, and is relatively more

voluminous than the parieto-frontal or "psycho-motor region;" that is, if the altered relations be due to unequal development of the contained organ and not of the containing cavity—a proviso that diminishes the significance of the observed facts. In adult life the relations between sutures and fissures continue invariable; we have seen that the occipital and temporo-sphenoidal lobes appear to maintain the same limits as the bones they lie beneath, and we might be tempted to presume a parallelism between a cerebral region and its cranial cover.

Amidon on the Effect of willed Muscular Movements on the Temperature of the Head: new Study of Cerebral Cortical Localization. (*New York Arch. of Med., April* 1880.)—Lombard, in 1867, was the first to investigate the relation between mental effort and cerebral, or rather suprajacent cranial, temperature; he employed a thermo-electric apparatus showing variations of less than $\frac{1}{1000}°$ F., and thus demonstrated a rise of temperature in the head and fall in the legs with various attractions and efforts of attention. Since that time the subject has been pursued, both as regards parts of the brain or suprajacent parts of the cranium, by a series of observers, Schiff, Broca, Gray, Maragliano, Séguin, &c.

The present paper, to which a prize has been awarded by the New York College of Surgeons and Physicians, is an outcome of the supposition that with the exercise of a peripheral group of muscles, heat generated in cortical centres might manifest itself to surface thermometers placed on the scalp; the writer relates the striking result of a preliminary experiment to test this view: "Surface thermometers were arranged in a strap passing across the vertex, where the centre of the arm was thought to be. After 15 m. the temperatures were recorded; violent flexion and extension of the forearm was then kept up for 10 m.; the temperatures now taken showed that a rise of more than 1° F. had taken place over the centre for the arm on the left side of the head only." The following are selected from the detailed accounts of further researches on the "thermic foci," made with due precautions by application of numerous surface thermometers to the scalp. "With movements of the whole upper extremity (flexion and extension of the fingers and forearm and some rotation of the shoulders) a rise of temperature varying from 2·5° to 2·75° F. was caused over rather a large area, extending on the median line from a point 14 cm. behind the root of the nose, about 10 cm. back on the same line,

laterally about 9 cm. from the median line." This area was further subdivided as follows: prolonged and repeated contraction of the biceps caused a rise greatest near the median line, about 19 cm. from the root of the nose; by further approximation of the thermometers, an area of maximum rise was defined 3 cm. long, 5 cm. broad, near the median line from a point 17 cm. to one 20 cm. behind the nose. The area of rise caused by triceps exertion is placed behind the preceding area of biceps exertion, near the median line, 3·5 cm. long, and 6 cm. broad. That caused by flexion and extension of the wrist and fingers is placed at about the same distance from the nose as that of biceps exertion, but further from the median line; in the diagram its upper border considerably overlaps the lower border of the biceps and triceps area. This and other overlappings are attributed to oblique radiation of heat, and to insufficient limitation of muscular movement. The anterior part of the above area adjacent to the biceps area is assigned to flexors; the posterior part adjacent to the triceps area, is assigned to extensors of the fingers and wrist. General movements of the shoulder-joint and scapula caused a rise over a large area, anterior to the area for the arm, approaching near to the supraorbital ridge in front, extending laterally 8 to 9 cm. Of this thermal area, 5 cm. long, 4 to 5 cm. broad, extending from the biceps area to within 13 cm. of the nose, is attributed to the deltoid exertion. To the trapezius and levator anguli scapulæ is attributed an area extending along the median line, anterior to the deltoid area, to within 5 to 6 cm. of the nose, and being 6 to 6·5 cm. broad. By exercise of the pectoralis an area of increased temperature was obtained external to the preceding, opposite to a point on the median line 14 cm. from the nose, being 3 cm. long, 3 to 4 cm. broad. An area of equal size behind the preceding is allotted to the latissimus dorsi. Movements of the head and neck were found to cause rise of temperature over a considerable area in the lateral frontal region external to the shoulder region. General movements of the leg and thigh caused an elevation of temperature over an indefinite area lying behind that of the upper extremity. Flexion of the foot with extension of the toes gave a rise in an area (anterior tibial) 4 cm. long 5 cm. wide, lying next the median and posterior to the area of the triceps brachii. Contraction of the calf-muscles caused rise in an area behind the preceding 2·5 long by 5 cm. broad. "Contraction of the quadriceps extensor femoris developed the rather startling fact that activity of this muscle caused a rise of temperature over a

correspondingly large area of the posterior part of the head." It occupied a position next the median line of the head, extending from the posterior boundary of the calf area 30 cm. behind the nose, very nearly to the occipital protuberance, 38 cm. behind the nose, averaging 5 cm. in breadth. "Some rises in this area amounted to 2·75° F." Other areæ, external to the preceding, are distributed to flexors of the thigh, abdominal muscles, erector spinæ. The thermal areæ of facial and lingual muscles are individually defined in the lateral region of the skull external to the centres of the upper extremity, 8 to 10 cm. above the external auditory meatus.

In conclusion the writer draws attention to the striking correspondence between these thermal areæ on the scalp and Ferrier's psychomotor areæ of the cortex, and asks whether some of the elevations referred by Lombard to mental states are not largely due to muscular action. He adds to Ferrier's chart by locating psychomotor centres on the frontal or psychical lobes, and remarks with some justice that the usual routine examination of a patient is not likely to reveal losses of power limited to muscles like the trapezius, deltoid, pectoralis, &c.; only when these symptoms have been properly looked for and not found, the belief in destructive frontal lesions without paralysis will be legitimated.

A. WALLER.

Visceral Neurology—*Respiration.*—Some of the most interesting contributions to visceral neurology during the last six months have been made in the nervous mechanism of respiration. Langendorff of Königsberg has published several important observations with respect to the situation and relations of the so-called respiratory centre. In the first place he has confirmed the view of Longet, Volkmann, and Schiff, that the respiratory centre in the medulla is double, either centre corresponding with its own half of the body, inasmuch as longitudinal division of the medulla in the middle line does not interfere with the rate or regularity of the respiratory movements. A connection between the two centres must exist, however, for it is a familiar fact that, when both vagi are cut, the respiratory movements continue to be synchronous. Langendorff's new observation is that when this direct connection between the two centres has been severed by longitudinal incision, section of either vagus causes disturbance of the respiratory movements on the corresponding side only, the rhythm becoming, as

usual, slower; whilst simultaneous section of both vagi leads similar disturbance on both sides, the rhythm on the two sides of the chest being different in time and frequency. Irritation of the central end of the divided vagus of either side, either directly, or indirectly (*e. g.* through the trigeminus in the nose) leads to arrest of the movements of the diaphragm on the corresponding side only. It thus appears that either vagus is connected with the respiratory centre of its own side, and that this, again, innervates its own half of the diaphragm; and, further, that beyond the direct local connection between the two centres, the cause of the correspondence in the rhythm and force of their action is to be found in their relation to the vagi. (*Centralbl. f. d. med. Wiss.* 1879, p. 913.)

In a more recent paper (*ibidem*, 1880, p. 97) Langendorff gives an account of certain experiments which appear to prove the existence of "respiratory centres" in the spinal cord. Both after section of the cord below the medulla, and after ligature of the cranial arteries (in the rabbit), he observed whole series of complete respiratory movements. The previous administration of moderate doses of strychnia remarkably increased the distinctness of these movements, which were not only spontaneous for a time, but also induced reflexly by stimulation of different sensory nerves. Langendorff suggests in explanation of these results that the automatic respiratory centres are situated in the cord, and that the medulla exercises only the function of a regulating apparatus, the importance of which in normal respiration must not be underrated.

Whilst the limits of the "respiratory centre" have thus been extended downwards, Christiani announces (*ibidem*, 1880, p. 273) that he has discovered a respiratory centre of limited extent in the floor of the third ventricle and in the substance of the optic thalami, beside the corpora quadrigemina. Irritation of this spot, directly or indirectly, causes respiratory arrest in inspiration. This observation is of great interest in connection with that of Drs. Martin and Booker, which was recorded by us in Vol. I. of 'BRAIN,' p. 585.

The influence of the vagus on respiration has long been familiar to physiologists; and attention has more recently been directed to the character and origin of the impressions which are being constantly transmitted upwards through it to the respiratory centre. The *Selbsteurung* theory of Breuer,—that the conditions of dilatation and contraction of the lungs, at the end of inspiration and of

expiration respectively, are stimulants to the immediately following acts, that is, to expiration and inspiration respectively; and that the impressions of the conditions of distension and contraction are conveyed upwards by the vagus to the respiratory centre—has been steadily receiving support from recent observations. Langendorff (De Bois Reymond's *Archiv f. Anat. u. Physiol.* 1879, Supplement-Band, p. 48) has discovered that when artificial inflation of the lungs is carefully carried out in *non-narcotised* animals, expiration is induced; and that dilatation of the lungs acts therefore as an expiratory stimulant. The experimenters who previously failed to obtain this result operated on *chloralised* animals. That the movements following inflation of the lungs were not the result of simple elastic recoil was proved by the fact that they were arrested by section of the vagi. On the other hand, Gad of Würtzburg, experimenting differently, doubts the truth of Breuer's beautiful theory as far as it applies to the inspiratory effect of expiration, inasmuch as interruption of the vagus does not increase but diminish the length of the expiratory act. Whilst making these experiments, Gad succeeded in finding a *non-irritant* means of suddenly paralysing the vagus, and thus of avoiding the stimulant effects of section, which always follow for a short time and disturb the results. This non-irritant interruption he found in freezing, which appears to cause no irritant effect whatever. (*Ibidem*, 1880, i. and ii. p. 1.)

Von *Anrep* (Pflüger's *Archiv*, xxi. (1 and 2), p. 78, has shown that the vagus of the cat possesses its familiar influence on respiration from the time of the birth of the animal.

The Heart.—The author just referred to, in the same paper, confirms to a great extent the observation of Soltmann that in kittens a few hours old it is impossible to stop either the whole heart or any of its parts by irritation of the vagus with moderate currents. In the course of two to seven days powerful currents arrest the ventricles but not the auricles. After seven to fourteen days the inhibitory centres are in a condition to cause stand-still of the whole heart. During the first days of life section of the vagus in the same animal has no effect on the frequency of the heart; and atropine causes no acceleration.

Three of the most prominent symptoms of exophthalmic goitre, namely, cardiac excitement, exophthalmos, and swelling of the thyroid, have been produced together experimentally in the rabbit by Filehne (*Centralbl. f. d. med. Wiss.* 1880, p. 192). This result was obtained by section of the restiform bodies in their anterior

fourth, without injury of the under surface of the medulla. The vagi thereby lost their control of the heart; the eyes frequently became prominent; and the thyroid occasionally swelled. Only in a single case, however, did the three phenomena occur together.

The Uterus.—Röhrig, experimenting on rabbits, has located the uterine centre in the cord in the lumbar and lower dorsal region (*Centralbl. f. d. med. Wiss.* 1879, p. 668). He has also found that the uterine contractions which follow diminution of oxygen or excess of carbonic acid in the blood, are directly referable to the condition of the cord. Anæsthetics appear to reduce the excitability of the uterine centre in the cord, whilst strychnia, picrotoxine, nicotine, coffee, and ammonia excite the cord and the uterus. Ammonia alone acts upon the uterus if the cord be previously destroyed.

J. Mitchell Bruce.

Buzzard on certain points in Tabes Dorsalis.—Three lectures have appeared (*Lancet*, January 10, February 14, May 1, 1880).

In Lecture I., after sketching the anatomy of the spinal cord, attention is directed to the degenerative changes observed in a case of transverse myelitis from Pott's disease. These extend upwards in the form of fasciculated sclerosis in the posterior median columns, and downwards in the posterior portion of each lateral column. A condition pathologically similar forms the essential change in tabes dorsalis, where it occupies the posterior root zones. The secondary degenerations are explained by Türck by reference to Waller's law in regard to degenerations of nerve-trunks after section. Buzzard thinks that there is clinical evidence to show that the change in tabes is not seldom also a secondary affection, and that it commences in the connective tissue. Duchenne's definition of the disease is followed, but Romberg's term, "Tabes dorsalis," is preferred to "Progressive locomotor ataxia." In a typical case exhibited, two symptoms are demonstrated which are not included in Duchenne's definition—the *absence of patellar tendon reflex* and *inaction or sluggish action of the pupils to light, whilst they contract normally during accommodation.*

In Lecture II. attention is given to the *pains* which, contrary to the experience of some observers, Buzzard finds present in tabes with very rare exception indeed. Graphic descriptions by patients illustrating the characteristics of these pains are quoted in large numbers. As regards their frequency Buzzard has analysed a

collection of fifty-four cases of tabes occurring in his own practice, and in only one instance were the pains entirely absent. Erb's experience, which is quoted, goes the same way, and out of a total of 110 cases analysed by these observers, pain was absent in only 7 cases, so that, as far as these figures go, pain is a symptom which occurs in tabes in the proportion of nearly 94 per cent. of the cases. Buzzard pictures, as very characteristic of early tabes, a man going about his usual occupation with activity, and presenting no sign of ill-health, but liable to have, from time to time, paroxysms of horribly severe pains, *not usually limited to the district of one nerve, as in neuralgia*, but commonly attacking more than one locality. Hints for avoiding errors in regard to the examination of the patellar tendon reflex are given; and it is pointed out that it is the absence of this reflex, when the quadriceps extensor muscle is in a normal condition as regards response to electric currents and blows, which is the striking feature in Westphal's test. A patient was exhibited, in whom this reflex was discovered to be absent six months after the first symptoms of tabes. As regards the frequency of the symptom, out of 79 cases of tabes in which this point was tested (Erb and Buzzard), in 76 the patellar tendon reflex was absent. In two out of the three exceptions to this rule of absence there was no ataxia of gait. Buzzard relates a case which well illustrates the value of the symptom. To his mind it holds the same rank as an objective symptom of tabes as is occupied by the characteristic pains amongst the subjective symptoms of the disease. These two symptoms are the most constant of all, and they are probably the earliest. Buzzard believes that if a patient presents both, no other is needed to form a diagnosis of tabes dorsalis.

Lecture III. deals with the oculo-pupillary symptom first dwelt upon by Argyll Robertson, and recently investigated extensively by Ch. Vincent in Charcot's clinique. Out of 51 tabetic patients in only 4 the pupils reacted normally. In 7 there was complete immobility of the pupils (usually combined with amaurosis), and in 40 the pupils did not react, or reacted imperfectly, to light; but contracted during accommodation. Buzzard has devised a very convenient method of testing the pupils for this condition. The patient is directed to look over the observer's shoulder at a distant object. A strong light is then cast upon the eye with the ophthalmoscopic mirror, and the pupil watched through a convex lens of about No. 12 behind it. If the pupil does not contract, the patient is next told to look alternately at the distant object and at one

within ten inches or so of his eye. Uniform illumination should be maintained, and the diameter of the pupil watched as the gaze is shifted. B. suggests that a very interesting generalization is derivable from this symptom. It is, he points out, the *reflex* movement of the iris which is abolished or greatly affected in tabes—the *voluntary* movement, that which occurs during accommodation of the eye for near objects, is preserved. This is precisely analogous to what happens with regard to the patellar tendon reflex, and the circumstance lends, he thinks, additional support to Pierret's view, that tabes essentially attacks the sensory side of the cerebro-spinal nervous system—reflex movements being excited by impulses travelling along afferent (sensory) nerves.

Next comes an investigation of the pains in the head in tabes; and it is shown that these are very common, and that their occurrence is readily explained (as Pierret describes), by imagining a continuation upwards of the sclerosis of the posterior root-zones (the essential change in tabes) to the descending root of the fifth nerve, which in the lower portion of the medulla oblongata forms a crescent enclosing the gelatinous substance of the posterior horn. An original and very clear diagram shows why lightning pains should be expected to occur in the region of the occipital and trigeminal nerves. Buzzard suggests that certain cases of supposed obstinate neuralgia of the fifth may really depend upon sclerosis, which is limited perhaps to this root. He describes some important diagnostic differences between cranial neuralgia and these cephalic pains of tabes. A patient was shown who had applied on account of excruciating pains in the head. He was blind. The symptoms suggested intracranial tumour, but the patellar tendon reflex was absent, and investigation showed that the case was one of tabes with cephalic pains and sclerosal atrophy of the optic discs. Another, who had optic atrophy and the Argyll Robertson pupil, was reported to stagger occasionally, but had nothing else except absence of patellar tendon reflex. The lecturer referred to another he had seen in private, in whom the absence of the reflex was the only symptom in addition to optic atrophy. There could be no doubt, he thought, that these were cases of tabes dorsalis as yet in a restricted form.

J. Hughlings-Jackson.

Abstracts of the Gulstonian Lectures on Epilepsy. By W. R. Gowers, M.D., F.R.C.P.

Lecture I. The subject selected for the lectures was a study of the history of chronic idiopathic convulsive diseases, founded on a

numerical analysis of a series of cases which had been under the lecturer's care, chiefly at the National Hospital for the Paralysed and Epileptic. All cases had been excluded in which there was any reason to suspect organic brain disease, and all cases of simple hysterical fits without distinct loss of consciousness. The cases included consisted of epilepsy proper, epileptiform convulsions, not due to organic brain disease, and cases such as are commonly designated hystero-epilepsy, which present, with loss of consciousness, convulsive movements consisting of more or less co-ordinated spasm, but often combined with distinct epileptic symptoms. Care was taken however to distinguish as far as possible the degree in which the various conclusions applied to one or the other class. The hystero-epileptic cases constituted 18½ per cent. of the whole (185 cases out of 1000 on which an accurate opinion could be formed of the character of the attacks).

The first lecture was devoted to the subject of causation. Regarding the influence of sex, the proportion of 1450 cases was found to be 53·4 per cent. females, 46·6 per cent. males, or nearly as 6 to 5. The excess of females was smaller, though still marked (52 per cent.), in the cases of pure epilepsy taken separately. Of the hystero-epileptics, females constituted two-thirds. The investigation of heredity was directed to the existence of epilepsy, insanity, chorea, hysteria, and certain forms of paralysis—paraplegia and infantile paralysis. Hemiplegia was excluded as primary vascular. The cases of hysteria or paralysis in relatives were insignificant in numbers. Inheritance was traceable in 36 per cent. (452 of 1250 cases), and in the same proportion in the epileptics and in hystero-epileptics. Heredity influences sex; in the cases with inheritance the females were 5 per cent. more numerous than in those without inheritance. The heredity was on the father's side in 35 per cent., and from the mother's side in 39 per cent., from both in 5 per cent., and only brothers or sisters suffered in 15 per cent. The side of the inheritance influences the sex of the sufferers. The percentage of males affected was 22 per cent. greater when the disease was inherited from the father's side than when from the mother's. The reason why the females are in such excess in the inherited cases is because the inheritance is more frequently maternal, and then more girls suffer than boys. A family history of epilepsy was obtained in three-quarters of the inherited cases. In half (240 cases) it existed alone; in 54, combined with insanity. Insanity occurred in a third (157 cases), and existed alone in the majority. Chorea occurred in relatives of 35 cases.

The mother is herself less frequently diseased than the father: she is as frequently epileptic as the father, but is much less frequently insane. A case was related in which 14 members of the family suffered from epilepsy.

Phthisis has been supposed by some to predispose to epilepsy, and is certainly frequent in the family histories of epileptics, but others believe not more so than is explained by its commonness. Individual cases suggest an influence, but statistics scarcely support it. A history of phthisis, in some relative, was obtained in 39 per cent. only, of 300 cases. The proportion of phthisis is just the same in those with and those without neurotic heredity, and the percentage of neurotic heredity is nearly the same in those with and those without a history of phthisis. Signs of inherited syphilis were present in 8 cases of apparently idiopathic epilepsy, and in 6 of these the fits began after childhood.

The influence of age was ascertained from 1450 cases, and the percentage commencing in decennial periods was—under ten, 29; between ten and twenty, 46; between twenty and thirty, 15·7; between thirty and forty, 6; between forty and fifty, 2; and over fifty, 3; 12½ per cent. of the total commenced in the first three years of life, and 5½ per cent. in the first year. The number, commencing per annum, fell to the sixth year, and then rose to a maximum in the sixteenth and seventeenth years, to fall rapidly after that age. The latest case was one which commenced at 71. Sex is influenced by age. In the cases commencing under ten years, the females exceeded the males by 6 per cent.; in those between ten and twenty, by 18 per cent.; between twenty and thirty, by 12 per cent. After this the relation is reversed. The males exceeded the females between thirty and forty years by 16 per cent.; between forty and fifty by 36 per cent.; between fifty and sixty by 40 per cent. Over fifty-five males only suffered. At each maximum period, both at infancy and puberty, the excess of females was very great.

The influence of heredity on age was ascertained from 1120 cases, and found to be much more uniform than current statements suggest. Of cases commencing under twenty (844 in number), 38 per cent. presented heredity. Of those between twenty and forty, 34 per cent.; and of those over forty, 27 per cent. The case which commenced at seventy-one presented heredity—his father having been epileptic. In the first twenty years of life the disease is hereditary in a larger proportion of females than of males; between thirty and forty, by more males than females.

How far do age and sex influence the occurrence of the hystero-epileptic or co-ordinated forms of convulsion? Of 1000 cases, this form constituted, in those commencing under ten, 15 per cent. of the male and 18 per cent. of the female cases. In those commencing between ten and twenty, it formed 14 per cent. of the male and 26 per cent. of the female cases. Between twenty and thirty the percentage of male hystero-epileptics is still 14, of females 21. Between thirty and forty the male cases fall to 12 per cent., the females rise to 26. In cases commencing over forty, this form is practically unknown in either sex. It is remarkable, however, that up to the fourth decade of life, one-third of the cases presenting hystero-epileptic phenomena occurred in males.

In considering the influence of *exciting* causes, the first point deserving attention is the cause of the large number of cases (180) which begin in the first three years of life. In one-third no causal information could be obtained. Of the remainder, in three quarters, the first fits occurred during dentition, and were referred to it. The percentage of neurotic heredity was 34, rather less than for the whole of life. If we ascribe to dentition convulsions the same proportion of the cases respecting which nothing was known, 7 per cent. of the whole become attributable to this cause. Teething convulsions are, however, well known to be usually associated with, and in large part the result of, the state of defective development which we call rickets, and the details of many of these cases supplied abundant confirmation of this. But in 27 other cases, which commenced after infancy, there had been severe dentition convulsions. If we attribute a predisposing influence to the early fits in these cases, we have a total of nearly 10 per cent. of the whole, in the causation of which rickets probably took part. This suggests that a considerable number of cases of epilepsy may be prevented by attention to the hygiene and diet of infants.

Excluding these cases, a probable exciting cause was ascertained in 438 out of 1150 cases, or 37 per cent., and was described in males (44 per cent.) more frequently than in females (31 per cent.), and the smaller proportion in the latter was chiefly due to the large proportion of cases in women between ten and forty years for which no exciting cause could be traced. In males the proportion presented but little variation in the different periods, increasing, however, late in life.

Of the cases presenting an exciting cause, more than a third (157 cases) were attributed to psychical influences, and of these fright caused no less than 119. It is effective chiefly in early life

—only 3 commenced after thirty, and 100 commenced under twenty. The age at which the two sexes suffered accords with their known emotionality—they were affected equally under ten. The males were to the females between ten and thirty as 3 to 4; between twenty and thirty, as 3 to 13; and over thirty, the only cases were in women. The influence of this cause is readily intelligible when the motor and visceral disturbance which alarm causes is considered. In one-third the first fit occurred immediately after the fright, in the others an interval elapsed. Two-thirds of the cases due to this cause were epileptic, one-third hystero-epileptic. The form of the fits is influenced by the interval after the fright—the shorter this is, the larger the proportion of cases of hystero-epilepsy. Other forms of mental excitement caused the first fit in 19 cases. Anxiety was the cause in 29 cases, in which males preponderated.

Sixty-five cases were apparently excited by blows and falls on the head. This cause affects the sexes equally under ten, but females after this date in gradually decreasing numbers. In two-thirds of the cases the injury was a fall, in one-third a blow. In one-fifth the first fit occurred instantly, in the others an interval elapsed. Exposure to the sun was the alleged cause in a considerable number of cases, and seemed probable in 27—20 males and 7 females. The males were distributed equally through life, most of the female cases commenced in childhood. The form of convulsion was almost always pure epilepsy. In 10 there was a distinct attack of sunstroke.

An acute disease was the cause of the first fit in 37 cases, 14 males and 23 females. In 9 it was measles, and in 19 scarlet fever. In many of the latter cases there was no dropsy or ear-disease; the first fit occurred during the height of the fever, or during convalescence, and was apparently due to the effect of the scarlet fever poison on the nerve-centres. In 9 cases the first fit was after immersion in the water, in 6 it was ascribed to digestive derangement, and in 6 to intestinal worms, the attacks continuing after the worms had been expelled. Chronic alcoholism caused 13 cases. Six patients were the subjects of chronic lead-poisoning. In 2 of these there was renal disease, in 1 fits, existing before, were intensified by the plumbism, in the remaining 3 the cases appeared to be saturnine epilepsy, chronic, resembling ordinary epilepsy. In 2 other cases chronic renal disease was the only discoverable cause of chronic epileptiform convulsions, without other signs of uræmia. Both lead and renal disease occasionally cause mental derangement. One case was apparently due to working in a tobacco factory; one commenced after the inhalation of chloroform, and in one,

attacks which had ceased for many years, recommenced after the inhalation of nitrous oxide.

Of sexual processes, retarded or absent menstruation coincided with the first fit in a large number of cases, in girls at puberty, but the causal relation between the two was difficult to ascertain. Masturbation is frequent in epileptic boys, but was certainly the cause of the fits in only a few cases.

The heart was not examined in all cases, but in 93 abnormality was found; frequency of action in 8, irregularity in 9, reduplication of the first sound in 13 (all young). In 30 cases there was valvular disease; mitral regurgitation in 20, mitral constriction in 7, aortic regurgitation in 3, aortic constriction in 1. There was dilatation only in 20, and hypertrophy only without renal disease in 2. In some cases the association with epilepsy seemed accidental; in some others the cardiac disturbance may have been due to the strain during the fits; in others the heart-disease had preceded the epilepsy, and may have been one of its causes.

In 20 cases of epilepsy the patients had also had chorea. In 8 the epilepsy existed first; in 12 the chorea preceded the epilepsy, in 4 immediately, in 5 at an interval of years. It seems probable that the disturbance of the motor centres during chorea may predispose to epilepsy.

Lecture II. Of the symptoms of the series of cases which formed the subjects of the lectures, the first point examined was time at which attacks occurred. Of 840 cases the fits occurred at night in one-fifth, in the day in rather more than two-fifths, and both night and day in rather less than two-fifths. In 5 per cent. they occurred only in the early mornings. There was no difference in these respects between the cases of pure epilepsy, and those with hystero-epileptic symptoms. An equal proportion occurred by night. The *first* fit occurred in epileptics rather more frequently by day than by night, in hystero-epileptics much more frequently by day. When the first fit occurred in the day, the subsequent fits occurred only in the day in half the cases; only in the night in one-seventh, both night and day in one-third. When the first fit occurred during the night, the subsequent fits occurred only at night in about two-fifths of the cases, both day and night also in about two-fifths, by night only in one-sixth. If fits which have recurred at night only, commence to occur in the day, they commonly continue at night. If they have existed only by day, and occur at night, they commonly cease by day. If they have

occurred both day and night, they often cease to occur in the day and continue at night, but very rarely cease at night and continue by day.

Of 82 cases in menstruating women, in one-twelfth no attacks occurred at the time of menstruation; in more than half they were worse at those periods, commonly before, or during, menstruation, rarely after.

Of the symptoms of the attacks, that selected for detailed analysis was their modes of onset. "Auræ" and "warnings" are now known to be the effect on the consciousness of the commencing change in the brain, and so give important information regarding the region of the brain on which the process of the fit begins. This mode of investigation was initiated by Dr. Hughlings-Jackson. The "discharge" may begin in an intellectual, motor, or sensory centre, and the distinction of the two latter is of great importance in their study.

Of 1000 cases, warnings were present in the attacks of 505, and the results of the analysis of the latter were described at length. The auræ may be referred to almost any part of the organism, but were divided, for clinical convenience, into seven groups.

(1.) Unilateral peripheral auræ (86 cases) were referred to one side of the tongue (7), face (17), arm (45), leg (15), and trunk (2 cases). No cases of presumably organic disease were included. The commencement on the tongue was less frequently by a movement than by a sensation. The normal associations of function were reproduced in certain combinations of the aura, viz., with movement in the lips and jaw, and with nausea. All cases commencing on the tongue were right-sided, which suggests (as does its frequent paralysis in aphasia), that its representation is chiefly on the left side of the brain. The attacks commenced in the face as frequently on one side as on the other, but much more frequently by a motion than by a sensation.

Commencement in the arm, when by movement (in eighteen cases) was almost always in the hand as a whole or arm as a whole; when by sensation (in fourteen cases) it was commonly in a definite part of the hand, and never higher than the wrist. In five other cases the sensation was probably motor: a sense of movement without movement. The attacks beginning by movement, or a motor sensation, commenced in one arm as often as in the other; those beginning by sensation began in the left hand twice as frequently as in the right. In cases of progress before loss of consciousness, the course was to the head, or down the side to the leg.

Commencement in the leg was by a movement (in five cases), usually in the foot, once in the hip; or by a sensation (five times), usually in a definite part of the foot. When the aura progressed before loss of consciousness, it passed up the side to the head, or down the arm. In these limb auræ there are two ways in which the second limb is affected, one by passage by continuity, up or down the trunk, and down the second limb; the other by fresh commencement in the extremity of the second limb, and passage of the aura up both. There is reason to believe that when the passage is by continuity through the trunk, the discharge in the sensory centre leads, and a case was related in which a sensory aura had this course, and motion was only added when it had gone down the second limb, *up* which the *spasm* passed. Special sense auræ were never associated with tongue, face, or leg auræ; and with an arm aura only when it began with sensation. The known relations of the cortical centres in the brain afford only a little help in explaining the progress of auræ; probably because this is largely influenced by the position of the undefined sensory centres for the limbs, in which many discharges seem to begin. Of the fits commencing unilaterally, 88 per cent. were purely epileptic, 12 per cent. presented hystero-epileptic symptoms.

In the second group were placed certain general auræ, bilateral sensations in the limbs, tremors, starts, malaise, &c. The commencement was in both arms in 11 (almost all purely epileptic), in the legs in 12 cases (3 certainly and 4 probably hystero-epileptic). Trunk auræ, not visceral, were rare, and were referred to the spine: the attacks were all purely epileptic. So also were the attacks commencing by general tremor (13 cases), and starts or jerkings (10 cases). Malaise constituted the warning in 15 cases, and faintness that of 13 cases; and of the attacks preceded by these general *sensations* about one-half were hystero-epileptic.

The third group comprehends auræ referred to certain viscera, mainly those to which the pneumo-gastric nerve is distributed; the epigastric sensations, choking, dyspnœa, nausea, and cardiac sensations. These were present in 106 cases. The epigastric aura (51 cases) consisted of actual pain in a third; it was sometimes referred to the left hypochondrium, but never to the right. When pain, it remained at the epigastrium until consciousness was lost. When a vague sensation, it often passed up to the head, or to the throat as choking. The latter sensation seems to be the same as the globus hystericus, and may be described in identical terms, even in organic disease. The warning was simply "choking,"

referred to the throat in eighteen cases, and dyspnœa or suffocation in six cases. These ascending auræ seem referable to the respiratory function of the vagus; the epigastric *pain* seems connected with the gastric functions, for in many cases nausea, and in some retching, was associated. Cardiac sensations constituted the warning in 16 cases; pain in 2, a vague sensation in 3, and palpitation in 11. Hystero-epileptic cases constituted one-fifth of those with an epigastric aura, or with chest dyspnœa, one half of those with a throat aura, and one-third of those with a cardiac aura.

The fourth group comprehends the auræ which consist in the fact of rotation, or the sense of giddiness, described as no less than 90 cases. Most were epileptic, one-sixth only being hystero-epileptic. In most, consciousness was very early lost.

Sensations referred to the head (fifth group) preceded the fits in 50 cases; vague in 29; pain in 21. In one-fourth of these cases the attacks were hystero-epileptic, and the proportion was nearly the same, whether the warning was a vague sensation or actual pain. Sudden somnolence was the warning in 5, and sudden inability to speak, in 9 cases.

A psychical aura (sixth group) was described in 25 cases; an emotion in 10; an idea in 15. The emotion was always fear, but bore no relation to fright as a cause. Associations with other auræ were rare; and in no case was it combined with an epigastric sensation. Only cases were reckoned in which the emotion was mentioned spontaneously by the patient, since, in most cases, with a deliberate aura, some alarm is felt. All the cases were purely epileptic. The intellectual aura was commonly a vague idea. Several patients described sudden recollection of many past events. A sudden sense of strangeness was felt in a few cases.

The last group consists of the auræ referred to the special senses (119 cases). An olfactory sensation, usually unpleasant, was noted in 7 cases, all purely epileptic, a gustatory sensation, described as a taste between sour and bitter, in one case only. In 84 cases, the warning was referred to the organ of vision. It was a sensation in the eyeball in 7; diplopia in 5. In a few there was an apparent magnification or diminution in the size of objects, probably connected with a commencing discharge, or inhibition, of the visual centre. Loss of sight preceded loss of consciousness in 26 cases (4 hystero-epileptic). In 46 cases the aura was a visual sensation (35 certainly purely epileptic). In 17, colours were seen, which could be described in 15. Red and blue were the only colours seen alone; one of them was always mentioned, and both

were present in half the cases. The order of frequency of colours was red (11), blue (8), green (3), yellow (3), purple (1). This order does not correspond with the order in the spectrum, nor with the size of the fields of vision, but it does with the visibility, of colours in direct sunlight according to Cohn, which is red, blue and green equal, yellow, violet. In 14 cases the aura was a highly specialized sensation, a visual idea, which in one case succeeded, in another coincided with, a simpler visual sensation; in the others, came alone and first, and must have been due, not to "loss of control" but to the discharge. Associated auræ were rare, but one instructive case was related at length; an epigastric sensation ascended through the chest as beating, and then was heard as knocking. This was followed by hissing, then by the vision of an old woman holding out a strongly smelling substance, then by two lights; lastly a sense of choking immediately preceded loss of consciousness. The only anatomical fact recorded regarding these auræ was a case, published by the lecturer, in which attacks preceded by a flash of light and micropsy were due to a tumour behind the angular gyrus.

Auditory warnings existed in 27 cases; sudden loss of hearing in 6; an auditory sensation in 21; a loud sensation of low specialisation, "crash," "blow," "hissing," &c., in 12; in others an "auditory idea." In several cases auditory and visual sensations were associated, and in some they were alike in character, *i.e.* in degree of specialisation. An unpublished case was mentioned, which the lecturer had watched with Dr. Hughlings-Jackson, in which attacks preceded by an auditory sensation referred to one ear were due to a tumour which had commenced in the white substance of the opposite temporo-sphenoidal lobe, within the convolution in which Ferrier places the auditory centre.

Of the other symptoms of attacks, time permitted mention of only one or two. Pallor of face is, it was urged, much less frequent than current statements suggest, and in both major and minor attacks may be entirely absent. The tonic and clonic spasm are continuous; the interruptions may be felt by the hand before they are visible to the eye. Slight attacks may consist of either clonic or tonic spasm: when the latter only, the attack is allied to hystero-epilepsy. The hands in an epileptic fit are not often clenched; more often they are in an interosseal position, the wrists flexed, the fingers flexed at the metacarpo-phalangeal, extended at the other points; the thumb bent in: an exaggeration of the position seen in tetany.

Some forms of hystero-epileptic attacks seen in this country were then described: Their characteristic is *co-ordinated* spasm, movements of quasi-purposive character. They have alliances with epilepsy and insanity as well as with hysteria in its simpler forms. In the first variety, resembling most that described by Charcot, the patient suddenly falls, and passes into a condition in which tonic spasm, often with outspread arms, alternates with violent movements, opisthotonos, wild movements of the limbs, &c. There is often internal strabismus, and frequently manifestations of intense terror. When severe, this frenzy and violence might be described as compressed mania. Sometimes there is a curious theiro-mimicry, and the patients will bark, and will bite in a strangely animal manner. In women ovarian tenderness sometimes exists, but the lecturer had never known ovarian pressure in the natives of this country succeed in bringing on these attacks, and, although it will sometimes arrest them, cutaneous faradisation does so more readily. Such attacks occur most commonly in girls and women, but also in boys and occasionally in adult men. A second variety is characterised by violent emprosthotonos, a third by curious rhythmical movements of flexion and extension of the legs, and a fourth by intense dyspnœa, reaching almost the point of suffocation. An instance of the latter, a somewhat rare form, was narrated at length.

Lecture III. The third lecture was devoted to pathology and treatment. In idiopathic epilepsy the facts of pathological anatomy are of most doubtful value. The problem of pathology resolves itself, in the main, into four questions: What is the seat of the "discharge" which causes the symptoms of the fit? Is the seat of the discharge the seat of the disease? How far does such "discharge" explain all the symptoms of the fit? What is the ultimate nature of the morbid state which causes the discharge? The word discharge was used, without theory, to signify the sudden liberation of nerve-force.

With regard to the first question, the seat of the discharge, there are two classes of facts. (1) There is a "convulsive centre" in the medulla, capable of giving rise by its action to general convulsions (Nothnagel). (2) Of all regional diseases, lesions of the cortex stand incomparably first as a cause of convulsion, and experiment demonstrates that convulsions may be produced by irritation of the motor region of the convolutions. But even the second set of facts alone scarcely warrant the conclusion that epilepsy is a cortical disease, because deeper motor structures, excitable from

the surface (Burdon-Sanderson), may be the parts which discharge primarily in convulsions, quite similar to those produced by experimental irritation. The affection of consciousness does not prove that the discharge begins in the highest centres, since discharges in lower centres may affect the centres above them as well as those below (Robertson), and in current theory an explanation of loss of consciousness is found in vaso-motor spasm. Thus neither pathology nor experiment enables us to exclude the lower centres, and the latest writer of a systematic account of the disease, Nothnagel, finds no reason to doubt that epilepsy is primarily a disease of the convulsive centre in the medulla oblongata.

The clinical study of modes of onset, however, gives us further help. In the cases (no inconsiderable number) in which the warning is a special sense aura, or psychical aura, we must conclude that the process of the fit begins in the special sense and intellectual centres, within the hemispheres. Thus the teaching of pathology is right in its indications, and we must conclude that in many fits the discharge commences in the cerebral hemispheres. If so, what facts are there to indicate that in epilepsy the fits ever do commence in the medulla? Such facts are, it must be confessed, scanty. The pneumogastric auræ, i. e., the visceral auræ referable to the respiratory and gastric centres, might be regarded as evidence that the convulsions depend on discharges in the medulla. But these centres are certainly (as Dr. Hughlings-Jackson has insisted) represented in the hemispheres; they are readily affected in emotional disturbance; and facts mentioned in the last lecture show that they may be easily disturbed in discharges unquestionably originating in the hemispheres. But we must not conclude, from the absence of direct evidence, that no fits commence in the medulla, and in a curious case under the care of Dr. Ramskill, the symptoms suggested this seat, for there was general powerlessness, and the attacks could be at any time brought on by passive movements of the trunk. The wide extent and varied character of the initial symptoms of fits points (as Dr. Hughlings-Jackson has urged) to their probable origin in any grey matter in which sensori-motor processes occur, but the evidence that they originate in the hemispheres is much more frequent and much stronger than that they commence lower down.

The question whether the seat of the discharge is the seat of the disease may be considered together with the third question, does the discharge which causes the convulsion explain all the symptoms of the attack? According to current theories vaso-motor

spasm is the cause of loss of consciousness, and some have added, of the motor discharge itself. But vaso-motor spasm is not *necessary* to explain the loss of consciousness, since the "discharge," as we have seen, may produce it. On what direct evidence has the vaso-motor theory been based? Mainly on the facts that there is pallor of the face in an epileptic fit, and that cerebral anæmia will cause convulsions. But, it was urged in the previous lecture, that pallor of face is often absent. Moreover we cannot infer from the state of surface-vessels the state of deeper vessels. Pallor of face, when it occurs, may be the effect of the cerebral discharge, not an indication of its cause. Between the fact that anæmia of brain will cause convulsions, and that vaso-motor spasm is the cause of any of the phenomena of epilepsy is an unbridged gulf, which is widened by every increase in our knowledge of the clinical history of the disease. Dr. Hughlings-Jackson's theory, that local vaso-motor spasm results from the commencing discharge and determines its spread, seems unneeded and opposed to the fact that functional activity of organs (and of the brain, Ferrier) causes dilatation of their vessels. Thus it is urged that all the phenomena of epilepsy may be explained by the instability of grey matter, and that there is no warrant or need to go farther in explaining. Epilepsy is thus regarded as without any uniform seat: as a disease of tissue.

What is the probable nature of the tissue-change? The "overaction" has been regarded as the indication of an increased functional and therefore nutritive activity (Hughlings-Jackson), or as really due to a general impairment of functional power and under-nutrition (especially by Dr. Radcliffe). Modern physiology seems to indicate the direction in which an answer is to be sought. The notion that there must be a resistance to action in nerve-cells, opposing the tendency to liberate energy, has underlain such expressions as "nerve tension," but has only lately been formulated, and promises to give clearer views of many physiological problems, and, as Dr. Ringer has shown, of many pathological problems also. The amount of latent energy in the brain, held in check, may be infinitely greater than any manifestations of it would suggest. The phenomena of epilepsy better accord with the notion of an instability of resistance than an excess of the energy-producing function. Thus the convulsions from loss of blood may be best explained, and so also may the arrest of a commencing fit by a peripheral impression. Once arrested (by raising resistance), it does not recur, as it would if dependent on primary overaction.

The internal resistance is probably a higher function than the transformation of force, and so an apparent over-action, an excessive liberation of energy, may be the result of imperfect nutrition.

The phenomena of hystero-epilepsy are, it was suggested, due to an instability and discharge of grey matter of different position and perhaps character. When succeeding an attack of epilepsy, major or minor, they have been regarded by Dr. Hughlings-Jackson as due merely to loss of control by the discharge of higher centres, on the ground that actual discharge can only itself cause incoordinate "brutal" effects. But the co-ordinated spasm of a hystero-epileptic fit *may* commence in the first movement of the fit; and the discharge of epilepsy sometimes, as in cases mentioned in the last lecture, may cause special-sense or intellectual symptoms of very high specialisation. In cases in which co-ordinated convulsion succeeds an epileptic fit, the hypothesis of loss of control seems scarcely adequate. It may be one element, but an instability of the lower centre seems essential; for why should attacks apparently quite similar be never followed by these symptoms? Automatic action commonly succeeds epilepsy, but, it was urged, may, in rare cases, occur without preceding epileptic symptoms. It was suggested, therefore, that all co-ordinated and automatic phenomena, often succeeding epileptic symptoms, may sometimes occur alone. In some cases they may be due only to loss of control, but they have commonly an anatomical basis in lessened stability of the centres on which they depend; that all *may* be due to a primary discharge of the unstable centre, but that the frequency with which they occur alone lessens as we ascend the scale of complexity. Automatic action is much rarer without preceding epileptic symptoms than are the "co-ordinated convulsions."

The clonic spasm of the later stage of an epileptic fit is continuous with the earlier tonic spasm, and may be due to the venous congestion, not exciting it, but interrupting and arresting the previous tonic spasm. Local slight epileptiform seizures commonly consist of clonic spasm only, and, as the discharge spreads and becomes intense, the spasm becomes tonic. The two are thus rather variations of the same form, than of different kind.

The treatment of epilepsy, it was remarked, is a subject on which numerical analysis gives little help, because so many patients, whose fits come under treatment, relapse when treatment is relinquished. The time available permitted little more than a statement of the remedies most useful in 562 cases, in which the effect of treatment was carefully noted. The results showed that,

while we must not rely *exclusively* on bromides, on these our chief trust must still be placed. The value and method of use of bromides, alone and in combination, of belladonna, digitalis, opium, Indian hemp, picrotoxin, &c., and the occasional value of borax, were then alluded to. After referring to the treatment of hystero-epilepsy, especially by apomorphia, the lecturer remarked, in conclusion, that although the condition of many epileptics was still gloomy enough, yet the present generation had witnessed an advance in the treatment of the disease equalled in, perhaps, no other branch of therapeutics. Thanks to the influence of one drug, the use of which in epilepsy was due wholly to Fellows of that College, hundreds of sufferers had been cured, and thousands were leading useful lives who would otherwise have been incapacitated by the disease. For all the victims of the disease, we might surely trust that the progress of the recent past is the dawn of a brighter day.

W. R. G.

On the Disease called Sturdy in Sheep, in its Relation to Cerebral Localisation.—In the January number of the *Journal of Anatomy and Physiology* I published an article with the above title, and in the present communication I propose to give a brief summary of those points on which I dwelt at some length in that paper. But to render my remarks clearer, it will be necessary that I first sketch in outline the disease called Sturdy, which is so named from its prominent symptom being a rotatory movement of a very peculiar character. It is common among sheep wherever they are herded together and tended by dogs, the latter point being of special importance, for the disease consists in the presence in the brain of the sheep of an hydatid, the cænurus cerebralis, which is a cystic or bladder-worm, and is the larval stage of the tænia cænurus, one of the six varieties of tape-worm infesting the dog.

This fact having been conclusively shown by Kuchenmeister, it is not difficult to trace the course of events in the case of a sheep-dog afflicted with tape-worm. He wanders about, distributing the ova of the tænia on the pastures, where they are swallowed by the sheep while grazing. When they have reached the animal's stomach, they are acted on by the gastric juice, their hard coverings are dissolved, and the small six-hooked embryos escape. By means of their hooks they bore their way into the blood-vessels and are carried to the brain, where they take up their abode, either from their having some predilection for that

organ, or because the peculiar cerebral circulation of the sheep, with its wonderful *rete mirabile*, favours their doing so. Having reached the brain, they make their way out of the blood-vessels, part with their hooks, and gradually acquire the bladder-worm state, varying in size from a pin's head to a walnut. After a time they assume the polycephalous condition, and an hydatid in this stage of development consists of a thin transparent bag or membrane, containing a clear fluid, and having a number of small whitish spots like eggs on its surface, disposed in regular lines. These are the heads of the hydatid, and I wish to direct special attention to the fact that they are *exsertile* or *protrusible*, a characteristic of some importance. An hydatid cyst of this nature may attain any size, but is not actually incorporated with the brain substance, being surrounded with areolar tissue. One other point connected with the history of this hydatid demands notice. It is this: that after a variable time it causes softening of the skull in its vicinity, the bony roof of the cranium disappearing both by reason of the pressure, and also probably from the protrusible heads of the hydatids not only removing the brain substance but also eating away the bone itself. This soft and yielding spot forms a guide to the situation of the hydatid.

Passing next to the symptoms which this malady gives rise to, we find that they of course vary with the stage which it has reached. Its earliest indications are those of cerebral congestion, as shown by redness of the eyes and dilatation of the pupils, together with a desire on the part of the animal affected to separate itself from the rest of the flock. It is also dull and listless in its movements, and in its desire for food. Any temporary improvement in these symptoms is soon followed by signs of increased nervous irritation, the creature being easily and groundlessly alarmed, and delighting to stand over running water, as if the murmuring sound caused by it had some soothing influence over it. In addition, there is difficulty in grazing, the head often turning to one side, to be soon followed by the rotatory movement which is characteristic of the disease. When once this is developed it goes on increasing in frequency to such an extent that the animal is unable to feed; and though eventually the movement may diminish in intensity, or even cease altogether, the creature becomes so exhausted and emaciated that at last it dies, unless operative interference should be undertaken for the removal of the cyst; but even this, as usually performed, is a somewhat fatal operation, owing to the admission of septic air, which sets up

putrefaction in the contents of the cyst, leading to inflammation and death. Such was the course of events in the first case of sturdy that came under my notice; and I fancy, from what I can learn, it is the chief cause of mortality in the cases submitted to operation.

Space does not allow me to go into the discussion of any of the individual cases of sturdy in their bearing on the theory of localisation of brain function, although some of them presented points of considerable interest. I must content myself with remarking only on one or two other points which the disease brings out, and chief among these is the cause of the rotatory movements which are so characteristic of the malady, and which also occur, as is well known, in animals that have been experimented on. In this latter case they are seen after very different operations, and Brown-Séquard has tabulated a number of the injuries that cause them, showing they are confined to no particular structure. This fact is also borne out by the disease of sturdy, for they declare themselves in that affection irrespective of the structure involved. Some explain them by the theory that they are due to spasms and paralysis of the muscles on the side towards which the movement takes place, and more recently in a clinical lecture, published in the *Lancet*, December 13, 1879, I find that Dr. Broadbent attributes their occurrence in animals to the association of the nerve-nuclei of the fore and hind limbs in connection with their mode of progression. But from my own observations in the cases of sturdy which I have seen, I am inclined to regard these movements as having another origin. I look upon them as caused by the presence of the hydatid itself in the brain, which so acts as a cerebral irritant that it sets up manifestations of nervous energy, interferes with the proper working of the general co-ordinating mechanism of the brain, and disturbs the delicate and sensitive apparatus of the nervous system. As a result of this, inhibitory influences are brought into play, which act on the will and also on the different senses, which are not only affected, but it may be thrown into abeyance. Now we know how completely this body of ours depends for its daily activity and movements on the integrity and healthy working of the channels through which travel the various impulses that invade our frame. No better illustration of this can be given than the extent to which ordinary locomotion depends on the integrity of sight. As Mayo long since put it, "we lean on our eyesight as on crutches." These remarks apply with equal if not greater force to animals, for we are aware that Longet and Flourens produced vertiginous movements in

pigeons by blinding or evacuating the humours of the eye. Struck by the fact that, as far as I had seen, the rotatory movement was always to the side on which the cyst was, I was led to look carefully into the state of vision, and I invariably found that when there was present this rotatory movement, there was impairment of sight on the opposite side of the body.

The conclusion then that I was led to draw was that while the presence of the hydatid in the brain sets in action nervous force, which the weakened will of the animal is unable to control, the *direction* of the movements is determined by the side on which vision is impaired. There seems to me further confirmation of this in the fact that when the disease becomes far advanced, and the sight in both eyes is affected, as is sometimes the case, the vertiginous movements cease. Upon these grounds this disease seems to me valuable, inasmuch as it affords us an explanation of the cause of the so-called "circus" movements seen after experimental operations, and also furnishes us with a clue as to the influences which determine the *direction* of these movements.

Lastly, I would direct attention to the following fact, to which I have already incidentally alluded, that the feasibility of removing the cyst must not be lost sight of. Apart from the procedure affording a good means of testing the value of antiseptic and non-antiseptic methods of treating wounds of the cranium, to the physiologist it would be still more valuable, for it would allow of his operating at definite stages of the disease, or whenever particular symptoms supervened. He might thus ascertain exactly the lesion present, and whether the symptoms observed were temporary or permanent. The only precautions necessary would be to wait until the softening of the skull gave some indications of the situation of the cyst, and to perform the operation with antiseptic precautions (Lister's method), so as to reduce to a minimum the chance of any subsequent putridity and inflammation, which might interfere with the observations made. In support of this suggestion I would only briefly allude to one case in which I removed the cyst by operative interference. In that case one of the chief symptoms was loss of vision in both eyes, but the animal was dull and lethargic to a degree, and reminded one of a creature deprived of its cerebral hemispheres. In a few days after operation it recovered its sight, and was restored to its usual health and habits of life, so that one could not have told that there had been anything wrong with it. When it was killed, some weeks after, I found that the cyst had occupied a place in the brain closely approximating

to Ferrier's centre for sight. Now had I not operated on it I should have regarded it as a case in which the visual centre had been destroyed, but the complete return of that sense after operation indicated that it was really only in abeyance. In other words, the organ of sight was temporarily involved, owing to the disturbance set up in the mechanism of the brain by the presence of such an irritant as we have seen that this hydatid is. This return of a function that seemed destroyed as soon as the source of irritation was removed, is only another instance of how careful we should be in drawing conclusions from experimental inquiries, a caution fully borne out by Goltz's numerous physiological experiments.

GEORGE T. BEATSON, B.A (Cantab.), M.D. (Edin.).

Tripier on Anæsthesia from Cortical Lesions. (*Revue mensuelle*, January and February 1880.)—In this memoir M. Tripier reviews the various explanations which have been given of the paretic or paralytic symptoms resulting from cortical lesions, and gives the details of some recent experiments he has made (in four dogs and one monkey), as well as several clinical cases in support of the thesis, that lesions strictly cortical cause not only motor symptoms but also defect of sensibility, principally, but not exclusively, tactile.

In his previous experiments, in which he had shown that dogs never really recover entirely from cortical lesions, and that the defect in mobility could always be reproduced by morphia, he had failed to detect any deficiency in sensation; but in the experiments here recorded, made with the special object of testing the sensibility, he finds that after lesion of the sigmoid gyrus, there is not merely paresis of the limbs, but also deficient reaction to cutaneous stimuli—pricking with a pin—on the affected side. This is most marked in the face and paw, but is more or less general, and may be observed immediately after the lesion, or even up to nine months afterwards.

He concludes from this that the sensibility is blunted or deficient.

He, however, opposes the views of Schiff, &c., that the defect in mobility is dependent on the deficient sensibility, and gives experimental evidence of the fact that division of the sensory nerves of the limb does not cause such motor disorders as result from cortical lesions. The motor symptoms are therefore the direct effect of the cortical lesions, but these are also combined with defect in sensibility, though the two are independent of each other.

The clinical cases adduced are cases of hemiplegia combined with hemianæsthesia, of greater or less degree, in which no other lesion was discernible post-mortem than cortical lesion, the ganglia and internal capsule being to all appearance intact. He is of opinion that there is no special sensory region in the cortex, but that the lesions which mainly cause hemiplegia (fronto-parietal), also cause hemianæsthesia. The latter, however, is more transient.

[M. Tripier's argumentation is not very conclusive. His clinical facts prove nothing. He might have cited cases with similar symptoms without discernible lesion in the cortex or elsewhere. It has been indisputably proved, in opposition to M. Tripier's views, that we may have most complete motor paralysis from cortical lesions, with full retention of sensibility both to tactile and painful stimuli.

Assuming the complete accuracy of M. Tripier's experimental data, they prove nothing beyond deficient reaction to stimuli. That this deficiency is on the sensory side is an inference which is not admissible until a clear discrimination can be made between psycho-motor and mere reflex reaction. Such a standard M. Tripier has not supplied, and to do so is no easy matter. An animal may not react to a cutaneous stimulus, not because it does not feel it, but because its volitional power is impaired. Even as M. Tripier's own data show, the reaction to a painful stimulus, such as would normally excite reflex movements, is as distinct on the one side as the other.]

Sommer on Diseases of the Cornu Ammonis (*Archiv für Psychiatrie.* Bd. x. Heft 3).—Sommer tabulates 90 cases, collected from various sources, in which the hippocampus was affected along with other cerebral regions, from which it is clear that epilepsy is frequently associated with degeneration in this region and its neighbourhood. He estimates the proportion at 30 per cent.

The lesions are divided into two principal forms: 1, the classic form, described first by Meynert, in which there is atrophy and sclerosis in one or both cornua; and 2, lesions of various kinds, such as softening, softening with redness, redness and punctiform hæmorrhages, pressure, &c. The proportions in which the various affections occur are stated in a tabular form. A detailed description is given of the microscopical characters of the degeneration which occurs.

Affections of the hippocampus in epileptics are associated in a

large number of cases with excentric sensory disturbances, indicating that the sensory centre is here, or in close relation with it.

Of the 90 cases analysed, 38 had sensory disturbances in the form of hyperæsthesia, paræsthesia, and anæsthesia.

Of the 52 negative cases, the records are either incomplete, or the conditions were such as to preclude any observations on this point.

Of the 38 positive cases, 27 relate to affections of common sensation; anæsthesia 7 times, hyperæsthesia 3 times, paræsthesia (formication, &c.), and actual hallucinations 15 times, and lastly 2 in which, owing to the mental condition, it could only be inferred. Frequently the common sensory disturbances were associated with illusions of hearing and sight, less frequently of smell and taste. Occasionally, however, these latter were alone affected without implication of common sensation.

Our author regards it as beyond doubt that there is a direct relation between degeneration of the hippocampus and the disturbance of common sensibility.

He next proceeds to the consideration of the experimental, anatomical, and other clinical evidence in favour of the view that the hippocampus is the centre of common sensation.

The first is furnished by Ferrier's experiments, which are briefly analysed.

The anatomical evidence is as yet incomplete. He refers to the researches of Meynert and others, in which it appears that there is a direct relation between this region and the sensory tracts of the spinal cord.

The clinical evidence is also as yet very deficient. There are only two cases on record, one of these even doubtful, which indicate the sensory function of the hippocampus.

The first of these, related by Chvostek (*Wien. med. Wochensch.* 1871, No. 37–39), was a case of typhus with delirium, headache, and other cerebral symptoms. This was in 1853. Diminution of the sensibility of the left arm and leg, with formication and slight paresis remained, and at a later period paralysis agitans showed itself on the left side. Death occurred from phthisis in 1871. Section showed atrophy and sclerosis of the right hippocampus and portion of the neighbouring cortex, without other cerebral lesion.

The second case related by Claus (*All. Zeitsch. für Psychiat.* 1878, p. 335), was a case of cerebro-spinal sclerosis, in which there were paræsthesia and impaired sensibility in the upper and lower extremity and trunk. On section, along with other scleroses, there was marked sclerosis in both hippocampi.

D. FERRIER.

BRAIN.

OCTOBER, 1880.

Original Articles.

OPTICAL ILLUSIONS OF MOTION.

BY SILVANUS P. THOMPSON, B.A., D.SC.
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THERE are frequent occasions of conflict between the receptive faculties of the senses and the reflective faculties of the intellect, occasions on which the mind, prejudging of the sensation received, assigns it to a non-existent cause. Of all the senses none is more frequently the seat of such deceptive judgments than that of sight; and in the science of Physiological Optics a very considerable share of attention is claimed by optical illusions. For the purposes of convenience, we may draw a distinction between these illusions, which are the direct result of certain properties or imperfections of the eye as an optical instrument, and those which arise from obliquities of judgment in interpreting the sensations optically impressed upon the retina of the eye. In practice, however, it is almost impossible to draw a hard-and-fast line between the two classes of illusions, almost all partaking of both characters. Thus, for example, it has lately been shown that we habitually draw geometrical forms too large in the horizontal dimension as compared with their vertical dimension; we draw oblate ellipses where we intend to draw circles; the explanation of this being that with our *two* eyes we really *see* spheres as oblate ellipses. Here is, in

fact, an illusion of pure association—yet based upon the facts of physical and physiological optics. So, again, certain inequalities in the curvature of the lenses of the eye, producing the optical defect of astigmatism, cause objects that are horizontal in position to form images at shorter (or longer as the case may be) distances from the eye than the images of vertical objects; the result being that, unless the defect is corrected by suitable lenses, vertical and horizontal objects (such as the bars of a window) do not appear to be at the same distance from the observer, though really equally remote. This would, at first sight, appear to be a purely physical illusion, and not psychological. Nevertheless, a little consideration will show that since our perception of distance is a psychological factor in the case, and that this perception is based in part upon the muscular sensations of adjustment of the lenses of the eye to exact focus, the illusion is one which has a psychological as well as a physical *raison d'être.* Again, take some illusions ordinarily supposed to be one purely of mental association: the common illusion of every day that the sun or moon when a few degrees from the horizon looks larger than when high in the sky, appears at first sight to be due simply to the fact that when the orb is near the horizon the distant objects upon that horizon whose size we know, or can judge of, appear relatively small, and the sun's disc relatively large—in fact, that the illusion is one purely of association of ideas. Nevertheless, when we look a little closer into the matter, we find that our simplest conceptions of angular or apparent magnitude are very closely bound up with, if not directly due to, the sensations of muscular fatigue in moving the eyeball or head so as to bring the successive parts of the object into the centre of vision.

Hence, although optical illusions are of many diverse kinds—illusions of colour, illusions of form, illusions of size, illusions of distance, illusions of solidity, and illusions of motion—they have all to be considered from the twofold standpoint, the purely optical and the psychological.

For some months the writer of this article was engaged upon a study of one set of optical illusions, namely, the illusions of *motion*, and a number of observations, collected at intervals

over several years, have been added by him to the stock of knowledge previously gleaned by Brewster, Wheatstone, Faraday, Plateau and others. Brewster made a number of observations in the early days of railways on the various illusions which can be found by watching objects from a moving train; Wheatstone investigated a curious case of apparent fluttering motion at the border of two brightly illuminated coloured surfaces—due probably to the attempt of the unachromatic eye to obtain fruitlessly a distinct focus of the border line between the unequally refrangible colours—known as the illusion of the "Fluttering Hearts;" Faraday investigated the illusions produced by intermittent views of moving objects, since developed in the Phenakistiscope and the Zoetrope, and kindred toys, and due to persistence of visual impressions. Brewster, moreover, drew attention to the existence of another class of illusions—illusions of subjective complementary motion—the typical case of which occurs also in railway travelling. After looking out of the window at the pebbles and other objects lying beside the line, as they pass before the eyes, let the eyes be closed suddenly, when there will at once be perceived an apparent motion in the opposite sense, undistinguishable forms and patches of light seeming to rush past the blank field. This was recorded by Sir David in 1848, and the phenomenon was referred by him to a subjective complementary motion going on simultaneously, and so causing a compensation of the impressions moving over the retina. A kindred phenomenon had been even earlier noted by R. Addams, who, in 1834, narrated how, after looking for some time at a waterfall and then at the waterworn-rocks immediately contiguous, he saw the rocky surface as if in motion upwards with an apparent velocity equal to that of the descending water. This he ascribed to an unconscious slipping of the inferior and superior recti muscles of the eyeballs, which he thought occurred while watching the falling water, and which he supposed to continue unconsciously after the gaze had been transferred to stationary objects. This explanation differs from the one offered by Brewster, namely, that there was a subjective *opposite* movement going on simultaneously, so causing a compensation of the impressions

moving over the retina. Brewster's hypothesis is, indeed, extremely vague, and is neither physical nor psychological in any exact sense. If understood physically, it means that there is actually motion in the retina itself, which is hardly conceivable, since the structure of the rods and cones almost precludes even any idea of vibration, or of propagation of waves of motion by vibration, much less any movements of them as a whole. And, if the explanation is intended as a psychological one, something further is needful before the principle of compensation here laid down could become intelligible.

The first experiments made by the writer of this article upon illusions of motion arose from a casual observation in 1876. He had been preparing, for the purpose of testing astigmatism, a set of concentric circles in black and white, such as those shown in Fig. 1. Happening to shake the sheet on

Fig. 1.

which the circles were drawn, he noticed an apparent motion of rotation to be set up. The illusion is easily produced by imparting to the pattern a slight motion of the same character as that adopted in *rinsing* out a pail, but with a very minute radius of motion. All the circles will appear to rotate with the same angular velocity as that imparted. Now undoubtedly

the persistence of visual impressions has a good deal to do with the production of this illusion, which, by the way, succeeds best when the circles make from two to four turns in a second, and when the radius of the imparted motion is equal to the thickness of one ring, so that each black or white band is displaced through a distance equal to its own width in all directions successively. Nevertheless, the persistence of visual impressions will not explain all the facts of this curious illusion: for, in the first place, it is found that for increasing distances from the eye the concentric rings must be made wider if the illusion is to succeed; there being apparently one particular magnitude of their images on the retinæ which favours the production of the illusion. Again, if two such "strobic circles" (as I have called them) are printed side by side on one card, that set of circles seems to turn most effectively at which the eye is *not* looking. On stopping the "rinsing motion" suddenly, there appears to be, for an instant, a reverse motion. Finally, if a set of circles is "rotated" while another set lies motionless within the field of view, the second set will appear to rotate when the first are "rotated" in

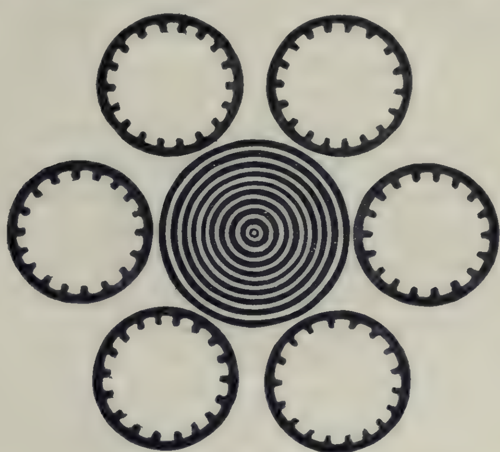

FIG. 2.

the manner described above. It is possible, also, to have a number of such apparent motions going on at once independently in one field of view. Fig. 2 shows a compound

pattern, containing an interior set of concentric circles and six internally-toothed wheels. When a very minute "rinsing" motion is imparted to this figure, the circles appear to whirl round while the toothed-wheels work slowly backwards, moving through one tooth while the circles whirl round once. Here again persistence of vision is concerned— but not exclusively.

Dr. Emile Javal, the able director of the Ophthalmological Laboratory of the Sorbonne, has recently advanced an explanation of these illusions different from that adopted by the writer, and in substance identical with that advanced by R. Addams in the case of the waterfall illusion. He avers that the eye, in order to observe a movement, follows the moving body for an instant and then suddenly slips back; that this oscillation, frequently repeated, is associated with a sensation of motion in the particular direction in question; and that when the eye is subsequently directed to a stationary object it continues the habit of thus oscillating, causing the observer to attribute to the object a velocity of opposite sign to that just observed. M. Javal alleges in support of this view the appearance presented in the ophthalmoscope of the retina of a person affected with *nystagmus.* This affection consists in continual rapid involuntary movements to and fro of the eye. The retina, under these circumstances, appears to be animated with a vibratory motion which M. Javal declares to be identical in character with the apparent movements of the circles. In another place, M. Javal has endeavoured to prove that the interior and exterior recti muscles of the eyeball are more prone to this slipping than are the superior and inferior recti, and that these illusions of complementary motion succeed better for motions in a horizontal sense than for vertical and oblique motions. My own experience, and that of other observers, admits of no such conclusion being drawn.

An experiment of Brewster's, which the writer tried without knowing at the time that Brewster had employed it,[1] has an important bearing on the muscular-slipping theory. A disc

[1] The same experiment was also tried by my friend J. Aitken, Esq., of Darroch, Falkirk, who independently observed the phenomenon described by Addams, and who has also communicated to the Royal Society of Edinburgh a number of experiments on kindred illusions.

marked out into black and white sectors, as in Fig. 3, was caused to rotate at about one revolution per second, so that the separate sensations of black and white were not confused. The eye was steadily directed for twenty or thirty seconds at the central point, and then the gaze was suddenly turned upon some fixed objects, or at a distant landscape. For two or three seconds a hazy rotation is noticed at the centre of the field of vision. Now if the muscular-slipping theory holds good, the complementary movement of rotation must be due to a slipping of the whole of the muscles of the eyeball, and would affect objects all over the field of vision with an equal

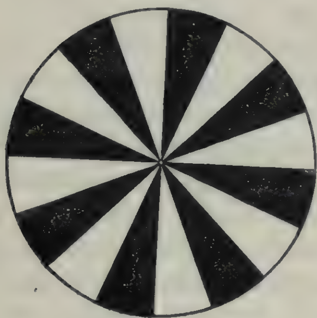

FIG. 3.

FIG. 4.

angular velocity. This is not the case, the apparent complementary rotation being confined to the central field, and with apparent angular velocities increasing toward the centre of vision. Furthermore, I have arranged two such discs so that they could be simultaneously in the field of view while rotating in opposite directions. When the gaze was directed first at a point between them and then at fixed objects, there appeared to be two portions of the field of view rotating, and animated with rotations in opposed senses. Clearly the eye cannot slip round in opposite directions at the same time. In all these illusions, moreover, it is found that this illusory complementary motion only occurs over limited parts of the field of view—namely, those which correspond to the portions of the retina which previously received the moving images. Thus, if a waterfall be looked at—as in Addams's observation—

the upward illusory after-motion is confined to a vertical streak across the field of vision. This fact alone is sufficient to negative the theory of muscular slip.

The final test to which I have appealed is, if possible, even more conclusive. It is probably a familiar observation that the end of the last carriage of a retreating railway train appears to shrink down smaller and smaller as it subtends a decreasing angular magnitude in the field of view. After looking at this motion for a sufficient number of seconds to fatigue the eye, stationary objects appear to be expanding. To produce this illusion more effectually, I take a disc like that shown in Fig. 4 (the figure is quarter actual size), marked out in spirals of white and black. If this is slowly rotated—say at about one revolution in two seconds—the whole pattern appears either to be running into, or running out of, the centre of the disc: there is a motion of convergence or divergence, according to the sense of the rotation. Let the disc be turned so as to cause an apparent convergence from all sides to the centre, and let the eye steadily watch the centre for about a minute, or until the fatigue becomes almost unendurable. Then look at any fixed object—the pattern of the wall-paper, or the dial of a clock—the object so regarded will for some two, or three, or more seconds, appear to be expanding from the centre outwards. The effect is still more startling if the object thus viewed be the face of a familiar friend. It is quite evident that the eyeball cannot slip in all directions at once.

I have therefore somewhat reluctantly been led to propound an explanation for these illusions, embodying the theory of them in an empirical law based upon the physical fact of retinal fatigue, and on the psychological fact of association of contrasts. It is as follows:—*The retina ceases to perceive as a motion a steady succession of images that pass over a particular region for a sufficient time to induce fatigue; and on a portion of the retina so affected, the image of a body not in motion appears by contrast to be moving in a complementary direction.* This law is precisely similar to that of the complementary subjective colours seen after fatiguing the retina by the image of a coloured body. Similar laws of physico-psychological after-

effects are abundant. A steady sound of one constant pitch ceases to be heard until we become aware of it by its cessation. A steady light of one colour, such as the yellow light of gas-flames, ceases to be noticed as a yellow light until some other colour-sensation break the illusion. The same is true of smells, of tastes, of the sensations of temperature, of the sensation of rotation after a waltz, and of many others. All these are probably only different instances of the operation of some much more general physico-psychological law. It is quite consonant with these kindred phenomena that when any region of the retina is affected by an image of objects moving steadily across the corresponding portion of the field of view in any given direction, that portion of the retina gradually loses consciousness of the motion, and perceives it only as a steady sensation, or as one of approximate rest. When, however, an object really at rest is looked at, the associative faculty seizes upon the contrast in the sensations affecting that region, and interprets the new sensation by imputing a motion in the opposite sense to the objects occupying the corresponding portion of the field of vision. I have proposed to give to the empirical law expressing these matters the name of the *law of subjective complementary motion.*

It is impossible to quit the subject without pointing out two lines of thought suggested by that which has been advanced.

Firstly, it is conceivable that the explanation here propounded may at some future time be superseded by a better hypothesis of a more purely physical character. Suppose, for example, that it could be shown—what I have reason to suspect, but have been foiled in all attempts to prove in any experimental fashion—that the eye has the power of altering at will the actual size of the retinal images by a double muscular adjustment between the magnifying power of the lenses of the eye and the distance of their equivalent optical centre from the surface of the retina, such a fact, once established, would entirely cut away the significance of my crucial test with the rotating spirals; and the apparent expansions and contractions of objects would be merely due to the continuous attempts of the eye to retain the retinal images of one constant size. If this were so (though I have failed in

every kind of attempt to devise some satisfactory test), it might also explain one little matter that is still very mysterious and unexplainable, namely, that in these illusions of expansion and contraction the changes of apparent magnitude often appear to take place by discontinuous jumps rather than by steady motions.

Secondly, it is found that these different illusions affect different individuals with very different degrees of success, some persons being much more sensitive than others to the after-workings of the subjective motion; and, indeed, there are individuals in whose case it is almost impossible to produce the illusions. Doubtless some of these differences may be accounted for by defects of vision, astigmatism, achromatopsy, myopy, and the like. But there is also a time-element in the case which varies very greatly with individuals, and even varies with the nervous states of the same individual. And this suggests the further thought that a careful comparison of individuals relatively to their illusion-capacity might elicit some interesting and perhaps valuable facts concerning the relation between the states of brain-organisation and the sensations of the more highly specialised organs of sense.

ON SOME POINTS IN THE DIAGNOSIS AND TREATMENT OF BRAIN DISEASE.

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It is a singular fact that, while pathological changes in certain areas of the cerebral substance invariably cause definite groups of symptoms, and can therefore, unless the patient succumb rapidly, be diagnosed with great certainty during life, on the other hand, coarse lesions may be discovered after death in important parts of the brain, without having manifested their presence by any apparent deviation from health during the patient's lifetime. There are those who contend that this latter event only occurs in slowly-growing tumours, which do not so much destroy the parts the sphere of which they invade as push them aside, and which increase so gradually in bulk as to give the structures concerned sufficient time to adapt themselves to altered circumstances, and to preserve their functions, however much their normal situation and nutrition may be interfered with. This opinion, however plausible at first sight, appears to us untenable, for we shall have to show that almost any morbid processes, such as hæmorrhage, softening, abscess, atrophy, tubercle, as well as the various forms of tumours habitually encountered in the brain-substance, such as glioma, sarcoma, osteoma, &c., may exist in important localities without giving rise to those special symptoms which we seem almost to have a right to expect under those circumstances. On the other hand, the reverse of this may be observed clinically in numerous cases, viz., that grave symptoms showing considerable alteration of brain-function exist, in which nevertheless coarse lesions are eventually shown to be absent, inasmuch as these symptoms yield completely to certain modes of

treatment which could not, with any show of reason, be supposed to have a curative influence upon such occurrences as hæmorrhage, softening, or tumour. Then again patients die occasionally with the symptoms of ingravescent apoplexy, and yet no hæmorrhage or other serious cerebral lesion is discovered on inspection.

By far the commonest form of brain disease to be met with in practice is hemiplegia, which comes on suddenly, affects the lower branches of the portio dura and the corresponding upper and lower extremity, is followed by late rigidity, and remains more or less permanent. In such cases we can say with a great degree of certainty that the opposite corpus striatum, and more particularly its internal capsule, must have been injured. It is true that a similar group of symptoms may be observed (*a.*) in disease of the upper portion of the pons Varolii; (*b.*) of the crus cerebri; (*c.*) of the centrum ovale; and (*d.*) of the central convolutions and the paracentral lobule of the cineritious substance of the hemispheres; yet we know by experience that hæmorrhage and softening, being the most frequent causes of the ordinary form of hemiplegia, have a special predilection for the corpus striatum, which receives its blood-supply chiefly from the lenticulo-striated arteries. These vessels, being the immediate continuation of the middle cerebral, and therefore the carotid artery, are more than others exposed to direct cardiac pressure, and therefore more liable to aneurismal dilatation and rupture. As embola are for the same reason more liable to be carried into them than into other vessels, necro-biotic softening from embolism is more frequent there than elsewhere; while thrombosis has no particular affinity to the vessels supplying the corpus striatum, and softening from thrombosis therefore occurs elsewhere as much as there.

With regard to hemiplegia from disease of the other brain-portions just mentioned, it is found that:

(*a.*) Lesions of the *pons Varolii* only rarely cause that form of hemiplegia of which we have just spoken, but almost invariably a motor paralysis which is known as alternate hemiplegia of the extremities and certain cranial nerves, more especially the sixth, the portio dura and the hypoglossus, which latter are paralysed on the side of the lesion, while the ex-

tremities are paralysed on the opposite side. Moreover anarthria or dysarthria is generally observed at the same time, and difficulty of deglutition, impairment of the circulation and respiration, and anæsthesia of the paralysed side of the face, *may* be found. The presence or absence of all, or some of these symptoms, will therefore enable us to pronounce for or against a pontine lesion.

(*b.*) Disease of the *crus cerebri* may cause crossed hemiplegia of the lower branches of the portio dura, the hypoglossus, and the corresponding upper and lower extremity; yet in some of these cases the fifth, and in more, the third nerve will be found paralysed, the latter suffering alternately with the other nerves, i.e. on the side of the lesion, while the other cranial nerves, together with the extremities, are paralysed on the side opposite to the lesion. Such unusual symptoms as third- and fifth-nerve paralysis, in connection with hemiplegia, are therefore quite sufficient to guide us in our diagnosis of such cases.

(*c.*) Disease of the *centrum ovale* cannot at present be diagnosed with any degree of probability. This structure may be extensively diseased without giving rise to any symptoms whatever; and where symptoms are present, we find crossed paralysis, either in the form of hemiplegia or monoplegia, leading us to suspect at first sight disease of the corpus striatum or certain portions of the cortex. Pitres has attached importance to the presence of unilateral convulsions, affecting the paralysed parts only, as distinctive of disease of the centrum ovale; but he has not succeeded in showing that in these cases there was not likewise irritation of certain portions of the cortical substance, which we know to give rise to this class of convulsions.

(*d.*) Disease of the *cortex* does, in exceptional cases, give rise to the ordinary form of hemiplegia such as we are in the habit of associating with corpus striatum disease; yet, where the antero-parietal or postero-parietal gyrus, or the paracentral lobule, or all these parts together are affected, the hemiplegia is generally "dissociated" or piecemeal; that is, we find paralysis of the arm and leg without any affection of cranial nerves; or paralysis of the arm alone; or of the arm and the

portio dura; or there is ptosis, and paralysis of the portio dura and hypoglossus, without affection of the extremities; besides which there may be certain forms of anæsthesia. Signs of motor irritation are the rule, either previous to, or after the occurrence of the paralysis, and appear as chronic spasms always affecting the same sets of muscles in one side of the face, or the corresponding extremities, and which often merge into general epileptiform convulsions.

Amongst those portions of the brain which may be found extensively diseased without there having been any symptoms during life, we would hardly expect to find the *medulla oblongata*, seeing the extreme physiological importance of the numerous centres which are there crowded together in a small compass. We know injury to the medulla to be immediately fatal; and any sudden interference with its structure, such as the rupture of a miliary aneurism and consequent effusion of blood into it, is likewise incompatible with life. Death is under those circumstances so sudden that one is inclined to attribute it to heart-disease ("apoplexie foudroyante" of the French authors). Such cases are very rare; and embolism of the vertebral artery leading to necro-biotic softening of the medulla is, for anatomical reasons, even more rare. On the other hand, thrombosis from disease of that blood-vessel is somewhat more frequent, but does not kill nearly with the same rapidity as hæmorrhage; for as the artery is only gradually occluded, the patient may survive for a few days, and present symptoms of apoplectiform bulbar paralysis, viz., dysarthria, dysphagia, paralysis of the soft palate and stertorous breathing, hoarseness or complete aphonia, hiccough, cough, vomiting, and irregular respiration and circulation leading to cyanosis. This cluster of symptoms coming on while consciousness remains intact, is highly characteristic for the lesion of which we have spoken. It is true that Charcot and Lépine have drawn attention to a somewhat similar concourse of symptoms arising from certain cortical lesions; but in the latter there is rarely dyspnœa or cyanosis, and *the symptom of aphonia occurs in no cerebral affection whatever except one of the medulla*, so that by this sign alone the diagnosis may be rendered certain.

While therefore in sudden lesions of the medulla, such as

hæmorrhage, or apoplectiform softening from thrombosis, symptoms of the utmost gravity are observed, we find, on the other hand, that this complex organ will apparently not resent disease coming on in a more insidious manner. Dr. Wilks has recorded the case of a girl who was admitted into Guy's Hospital, complaining of occipital headache. There being no other symptoms, the pain was thought functional, possibly hysterical. Two days afterwards she was found dying, respiration having suddenly become arrested. As the heart's action continued, artificial respiration was had recourse to for eight hours; and the heart continued to beat for twenty-five minutes after the cessation of these therapeutical efforts. At the post-mortem examination a glioma was found in the medulla infiltrating and encroaching on all the important centres, including the fourth ventricle. Other cases in which there was an almost entire absence of symptoms, have been seen by Ollivier, Louis, and Schulz. In the latter's, there was a glioma occupying almost the entire thickness of the medulla, of which only a small zone of nervous matter remained. There was also sclerosis of the lateral columns of the spinal cord, which during life had caused the symptoms of spastic paralysis; but no sign referable to the medullary disease had been noticed.

Next in vital importance to the medulla oblongata comes the *pons Varolii*, disease of which is much more frequent than that of the medulla. Hæmorrhage is almost invariably fatal, but not with such extreme suddenness as when the effusion takes place into the medulla, two or three hours generally elapsing between the first onset of the symptoms and death; yet where the clot is large, and presses considerably on the medulla, the fatal issue may be more sudden. Embolism of the basilar artery, which would lead to necro-biotic softening of the pons, does not occur for anatomical reasons; while thrombosis of that vessel, more especially in syphilitic subjects, is by no means rare. According to the extent of the thrombosis the symptoms may vary considerably, which need not surprise us, seeing the number of centres and conducting paths which exist in such abundance in this most complex structure. There are no cases of hæmorrhage or softening on record in which not some at least of the symptoms of alternate paralysis were present, viz.,

crossed hemiplegia of the limbs, and equilateral paralysis of the mouth, divergent strabismus, and anæsthesia of one side of the face, as well as dysarthria.

It is, however, different with tumours which, if growing very slowly, may become developed without causing any symptoms, and even gradually attain a considerable size. Such cases have been recorded by Hallopeau, Laborde, Henoch, and others. In Laborde's case there was a tubercle of the size of a filbert in the centre of the pons; and in Hallopeau's there was an area of tubercular disease in the central portion of the organ, which measured 2½ centimètres in its largest diameter. These cases are however exceptional, since generally speaking the symptoms in pontine tumours are of a most varied description, consisting in the sphere of motion of paralysis and convulsions, muscular rigidity, lock-jaw, nystagmus, pendulum-like movements of arm and leg; in the sphere of sensation of pain and of anæsthesia, which affects the face and the extremities, the affection being crossed in the latter; to which are sometimes added certain vaso-motor and trophic phenomena.

There is no portion of the brain where the peculiarity which we are considering obtains so frequently and with all different kinds of disease as the *cerebellum*, a circumstance which renders the diagnosis of cerebellar affections a matter of peculiar difficulty, and sometimes one of impossibility. Not only tumours, but hæmorrhage, softening, abscess, atrophy and tubercle, have been discovered by such observers as Bourneville, Nothnagel, Heusinger, and others, in which during life absolutely no symptom was noticed. In Ebstein's case, almost the entire left hemisphere was destroyed by an osteoma; in Lallement's, there was complete atrophy of the left lateral lobe without tumour, and also atrophy of the right olivary body, and part of the right corpus striatum; in Heusinger's, there was an abscess in the right lateral lobe which was apparently four or five months old; in Nothnagel's, a cicatrix in the corpus dentatum, which was evidently the remains of old hæmorrhage, the patient having eventually died of dysentery; in Mosler's case, an area of inflammatory softening in the nucleus dentatus of the left lateral lobe of the size of a pigeon's egg, the patient having suffered from diabetes and died of pleurisy without any symp-

toms on the part of the nervous system; while in Andral's case (that of a boy who died of pulmonary consumption) four tubercles in the left lateral lobe were found, three of them being of the size of a cherry and one of a filbert. Instances like these might be multiplied indefinitely, while in others again a variety of symptoms has been observed. As a general result it may be stated (*a.*) that there are no symptoms where the disease is localised in one lateral lobe, and where it does not produce pressure on neighbouring parts, such as the middle lobe, the pons Varolii, the medulla, or the corpora quadrigemina; and disease of one of the lateral lobes is therefore as a rule impossible to recognise during life; (*b.*) that symptoms of a definite character occur almost invariably where the middle lobe is affected; (*c.*) that the principal symptoms are the "reeling gait" (cerebellar ataxy) and a peculiar form of vertigo; and (*d.*) that all other symptoms which have been noticed in cerebellar disease, such as optic neuritis, vomiting, certain forms of paralysis and convulsions, nystagmus, headache, various kinds of anæsthesia, paræsthesia, and hyperæsthesia, deafness, and symptoms of mental disturbance, are uncertain and inconstant, and generally owing to pressure of tumours on distant parts, or to co-existing disease in other organs.

Of the *crura cerebelli*, only those which connect the organ with the pons Varolii—the middle crura—seem to give rise to special symptoms when diseased, while the inferior crura which connect the cerebellum with the medulla, and the superior ones which link it with the optic lobes, may become diseased without apparently resenting it. Again, simple destruction of the middle crura, as by hæmorrhage or softening, may pass unperceived, while irritation of these parts, chiefly by growing tumours, causes characteristic appearances, provided the connection between the crura and the cerebellum be not completely destroyed. These consist of certain forced movements or positions of the body, head or eyes, over which the patient himself has no control whatever, and which, if disturbed by others, at once reassert themselves. They occur sometimes in a state of unconsciousness; and where the patient is conscious, resistance on his part to such forced movements or positions is unavailing. Sometimes they appear in the form of fits, so that

the body is turned right over in bed four or five times consecutively, by the action of the spinal muscles, the patient occasionally resisting such movements by taking hold of the bed-clothes, but without effect. In other cases the patient remains constantly lying on one side, and if his position be shifted by the nurse, returns at once to the one previously assumed. Conjugate deviation of the head and eyes may also occur, or, as in Nonat's case, one eye may be turned outwards and downwards, while the other is directed inwards and upwards. The presence or absence of coma is of some importance in such cases; for conjugate deviation of the head and eyes is known to occur in hæmorrhage of the central ganglia, pons Varolii, and the meninges, together with coma, while in disease of the middle crura it may occur without coma.

The *crura cerebri* are, in their progress from the anterior border of the pons Varolii towards the central ganglia, often implicated in disease of the latter, yet may suffer by themselves of hæmorrhage, softening, &c., owing to disease of the external posterior optic arteries (Duret). As a rule, the symptoms are well marked, there being either crossed paralysis of the extremities, the portio dura, and the hypoglossus; or these symptoms are combined with equilateral paralysis of the third nerve, thus constituting a form of alternate paralysis which is peculiar, and from which a definite conclusion as to localisation in the crus may be drawn; while, of course, simple crossed paralysis of the extremities, the portio dura and the hypoglossus, may be due to a variety of other lesions. The paralysis of the third nerve may be complete, when there is ptosis, external strabismus, and diplopia; or it may be incomplete. In either case, however, the lesion appears to be seated in the internal portion of the crus, while affection of its external portion is more apt to cause anæsthesia than paralysis. No local symptoms were observed in a case of Gintrac's, where a tubercle of considerable size was found in the right crus of a boy six months of age; and of Delasiauve's, where an encephaloid growth was discovered in the left crus.

Localised softening and hæmorrhage do not appear to occur in the *corpora quadrigemina* or *optic lobes*, no doubt owing to their deriving their blood-supply from the posterior cerebral

arteries, which also nourish the optic thalami and other neighbouring parts, so that the latter are apt to be affected simultaneously. Griesinger taught that, when vision was normal, there could be no affection of the optic lobes; yet cases have been recorded by Kohts, Gowers, and Steffen, in which destructive lesions of these parts were unaccompanied by blindness. The whole subject is still involved in much obscurity, inasmuch as tumours may, simply by causing an increase of intra-cranial pressure, give rise to the choked disc, optic neuritis, and other symptoms, which have nothing whatever to do with their localisation in the optic lobes. Yet it appears probable that disease of the posterior portion, or the nates, is less likely to cause amaurosis than lesions of the anterior portion, or the testes; while on the other hand, palsies of certain branches of both third nerves seem to speak for affection of the nates.

If there be obscurity in the pathology of the optic lobes, Cimmerian darkness may be said to prevail in the sphere of the optic thalami, concerning which the best observers are at this moment in a state of apparently hopeless discrepancy. This is no doubt owing to the circumstance that truly localised lesions, whether from hæmorrhage, softening, or tumour, are very rare in these parts, and that in most cases which come under observation, extensive pathological alterations co-exist in important neighbouring structures. In consequence of this, the most fundamental points are as yet undecided, such, for instance, as whether the optic thalami have anything to do with the sense of sight or not, whether they are centres of common sensation or not, whether lesions in them give rise to the ordinary form of hemiplegia or not, &c. One fact, however, is certain, viz., *that hæmorrhage may occur in these parts without giving rise to any symptoms at all;* and Nothnagel has recorded a case in which there was even bilateral hæmorrhage in the thalami without the least suspicion of cerebral disease having been excited during life. This also holds good for tumours, as in Gintrac's case there was tubercle in both thalami in a patient who had never shown symptoms of brain-affection.

With regard to paralysis, it may be said that its presence speaks rather against, than for, disease of the thalamus.

Complete destruction of its posterior third has been seen without a trace of paralysis; in lesions of the middle third, paralysis has only been noticed when the area of disease was very extensive, and may therefore be inferred to have exercised pressure on the internal capsule, while where the area was small, no paralysis was present; and finally, disease of its anterior third is so constantly associated with destruction of the corpus striatum, and more especially the internal capsule, that the hemiplegia which is present in such cases, has to be referred to the latter structures. Common sensation was formerly believed to centre in the thalamus and to suffer from its destruction, but this theory has, for the present at least, been disposed of by the researches of Lafforgue. On the other hand, Crichton Browne has rendered it probable that disease of the thalamus causes diminution or abolition of reflex excitability, more especially in the upper extremity; and finally, certain forms of motor irritation, such as hemichorea, athetosis, and tremor, seem to be connected with disease in this part. Nevertheless we may say that the best-informed physician cannot at present diagnose disease of the thalamus with any degree of certainty.

(*To be continued.*)

RECURRENT HEADACHES IN CHILDREN.

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HEADACHES are of such common occurrence in young people that no physician whose observations are made largely among children, can fail to meet with many cases in which headache is the main subject of complaint. In dealing with this group of cases from a scientific point of view, it is evidently necessary to define the physical conditions which may be taken as signs of that mobile or irritable condition of the nervous system in which headaches are so common.

The study of diseased conditions of the brain has been greatly advanced by observing the conditions of the muscles of the face, eyes, limbs, trunk, &c. It is by the clonic muscular twitchings in chorea that we know the brain is choreic; it is by the condition of hemiplegia we are led to infer that there is a lesion in the basic ganglia of the opposite hemisphere; it is by the observation of labio-glosso-laryngeal paralysis that we diagnose disease of the olivary body. If, then, we would conduct our observations in the case of children who suffer from recurrent headaches on principles and lines of investigation similar to those which have led to important results in the study of chorea, hemiplegia, and other cases of organic disease, we must carefully observe the condition of the muscular system in such patients. Following upon these lines of argument, I have in many cases examined and recorded the conditions of muscles supplied by cranial nerves, and those of the upper extremities.

It is a matter of common observation that a patient may "look as if he had a headache."

Such an appearance of the face can only result from its colour, form, or mobile conditions, for these are its only distinguishing characteristics.

In children it is not uncommon to have "the expression of headache in the face;" so frequently was it observed, that I came to look upon it as a physical sign of the condition which produces headache.

A face may be analysed as to the three properties spoken of, and that as to either half of the face, or as to the upper, middle, or lower portions. If either vertical half of the face be covered in turn by a paper, "the expression of headache" is seen equally on each side, proving that the cause of the expression is bilaterally symmetrical. If the paper be held with one margin horizontal it may be made to cover in succession, (1) the forehead down to the level of the eyebrows; (2) the parts about the eyes from the eyebrows to the lower margins of the orbit; (3) the parts from the lower margin of the orbits downwards, including the mouth, cheeks, and alæ nasi.

In analysing faces to see wherein lay "the expression of headache," I looked at the faces of adults, and the expression seemed retained as long as the parts about the eyes were seen and lost when the middle zone was covered, i.e., this peculiar facies appeared due to the condition of the parts about the orbicularis oculi. There seemed to be a loss of tone in this muscle; there was an appearance of fulness and flabbiness about the lower eyelid; the skin hung too loose, and in place of falling against the lower eyelid neatly, as a convex surface, it fell more or less in a plane from the ciliary margin to the lower margin of the orbit. This condition is often seen best by looking at the patient's face in profile. This condition of the parts about the eye may often be seen in children, unaccompanied by any general change in the skin of the face, such as the flabbiness seen in emphysema, and the loose inelastic skin of senile decay. The expression is not due to local œdema, for it does not pit, and may often be removed instantly by causing the expression of joyousness or laughter, when the orbicular muscle is energised and temporarily recovers its tone.

More might be said of this antithesis of the muscular state in the opposite conditions of the centres of feeling. It is not suggested that the appearance of the face accompanies headache only, it is common in many conditions of depression.

Something might also be added of the partial ptosis and pigmentation of the lower eyelid sometimes seen.

The muscular condition of the hand in such patients seems to me equally characteristic with that of the face. There is a nervous condition of the hand, as there is a nervous condition of the face, and each can be described in anatomical terms. As the child stands up, holding his hands out before him on a level with the shoulders, palms downwards, and fingers spread out, there may often be seen a peculiar and, I think, characteristic condition of the hand. The wrist droops slightly, at the same time the phalanges are extended backwards upon the metacarpus, the second phalanges being slightly flexed, and the ungual phalanx either flexed or in a straight line with the middle phalanx. The thumb is usually simply extended backwards and somewhat abducted from the fingers. I think that this position of the hand may be taken as an item of evidence that the child is of a neurotic habit, with mobile nerve-centres; it may be seen in girls convalescent from chorea, and in others who are weak and nervous. This "nervous hand" is sometimes seen more marked on the left than on the right side. I have now under observation a lad and a man, both the subjects of partial hemiplegia apparently from organic disease, in whom this "nervous hand" is well seen on the weak side. Twitchings of the fingers were very commonly seen, the movements being usually of independent digits, and most commonly either lateral or flexor, less commonly extensor; the lateral movements appeared the most characteristic.

Believing that "the nervous hand" was owing to the condition of its muscles, and that the condition of its muscles was owing to the condition of certain nerve-centres, the conclusion arrived at was that the position of the hand must indicate the condition of those nerve-centres.

Considering the illustrations that Darwin has brought forward to prove that the emotions are expressed by the exercise of certain muscles, and that in opposite emotional conditions (i.e. opposite conditions of nerve-centres?) the antithesis of the emotional condition is expressed by an antithesis of the muscular condition, my observations were directed by this principle of antithesis.

A hand almost the antithesis of "the nervous hand," and

expressive of force and energy, is often seen in men when speaking earnestly. Here the wrist is extended, the metacarpo-phalangeal joints and interdigital joints are flexed, the thumb is also moderately flexed.

It is of course the 7th cranial nerve which connects the facial muscles with their nerve-centres. If the centres of the 7th nerve are disturbed in conditions of depression it is reasonable to inquire if the muscles supplied by other cranial motor nerves show any signs of relaxation or disturbed equilibrium.

The 3rd, 4th, and 6th nerves supply the eye muscles. Evidence of disturbance of the motor division of the 5th nerve is seen in the great frequency of tooth-grinding. In a large proportion of my cases, the teeth were found flattened upon their edges, as the result of tooth-grinding. This condition may, I think, be taken as another physical sign of the nervous condition. We know that the sensory branches of the 5th nerve are largely distributed to the meninges.

The tongue is supplied by the 9th nerve, and this when protruded was usually very unsteady, but not distinctly jerked in and out as in chorea.

There appears to be some evidence of disturbance of the pneumogastric nerve in the varying condition of the appetite, which is often voracious, often very poor; vomiting may accompany or succeed the headaches. Illusions of sight with headaches, such as sparks, colours, hemiopia, &c., are in my experience much more rare in children than in adults.

I found that very commonly the urine was of high specific gravity, reaching up to 1030 or 1035, and loaded with urea. Uro-hæmatin was also usually abundant; in both these facts these children correspond with those suffering from chorea.

Fifty-eight cases were arranged in a tabular form, as follows, showing the relations to age and sex:—

Ages.	3–4	4–5	5–6	6–7	7–8	8–9	9–10	10–11	11–12	12–13	13–15
Males 25	1	2	2	8	2	1	2	2	4	1	0
Females 33	0	2	3	1	2	5	5	4	2	4	5
Totals 58	1	4	5	9	4	6	7	6	6	5	5

Heredity produced a marked predisposition to this neurotic condition; in the 58 cases there was a history of recurrent headaches in the mother in 24 cases, in the father in eight cases, while in three cases there were examples of insanity in the family. In looking at the faces and heads of children suffering from headaches, I found in many cases signs of rickets, and when in the general clinique of children the patient was seen to be rickety, headaches were found in most instances; it seems then that rickets in childhood is a strongly predisposing cause of recurrent headaches.

METHODS OF PREPARING, DEMONSTRATING, AND EXAMINING CEREBRAL STRUCTURE IN HEALTH AND DISEASE.

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I.

COARSE EXAMINATION OF THE BRAIN AND ITS MEMBRANES.

IMPORTANT and necessary as are the minute investigations made by microscopic agency into the normal structure of the brain or its pathological deviations, we must guard ourselves from the very serious error of considering this method as essentially and exclusively necessary in these studies. The error to which I allude is a common one, and is too apparent to need much comment upon my part; for all who are engaged in the prosecution of cerebral histology must have recognised the prevailing tendency to disregard the naked-eye appearances of the brain, or to regard them as of very secondary import compared with a minute investigation aided by the complex armamentarium of the microscopist. If any of my readers are possessed of this notion I would ask them at the outset to rid themselves immediately of the fallacious idea which, if fostered, must prove a serious obstruction to the acquirement of that intimate acquaintance with the true significance of the varied appearances presented by normal and diseased tissues. The skilled obstetrician recognises as an invaluable acquirement that *tactus eruditus* which a constant and intelligent employment of a special sense can alone confer; and no less should the histologist endeavour to obtain a special visual tact, a highly refined and educated visual power which can alone enable him to recognise by the unaided eye appearances which pass wholly unnoticed by the casual observer. I cannot too strongly insist upon this point, for he who would successfully study the morbid anatomy of the brain must, as in

the morbid anatomy of other tissues, begin seriously to educate the eye to the coarse appearances presented to the unaided vision. The employment of a hand-lens of two to four inches focal length will prove of service here, the naked-eye appearance being contrasted with the magnified field, and the eye thus educated up to recognising characters which without the aid of the lens were previously indefinite or unrecognisable. Nothing beyond repeated and energetic efforts in this direction will enlarge the area of the visual field and present to the mind the manifold appearances which constitute an unbroken whole, and which are absolutely necessary to a refined interpretation of the picture presented to the mind's eye. Repeatedly have I had occasion to observe that the student, after a full curriculum of hospital training—a training which should pre-eminently involve the high culture of the sense of sight, hearing, and touch—fails wholly to appreciate the most obvious abnormalities of the cerebro-spinal tissues, and this because he has neglected to tutor the eye so far as to learn what to look for, and how to look for it. If you place before him the brain of a case of chronic mania, without coarse lesion, and note his observations upon the appearances of a section across the hemisphere, it will be found that beyond a statement that the grey matter is pale, anæmic, and wasted; that the white matter is altered in consistence, and presents numerous coarse vessels, his untutored eye teaches him no more; and should he be examined even for the grounds upon which these statements are made, a too evident vagueness will be apparent —his ideas on relative depth of cortex or anæmic states of the brain and consistence of its tissue are gauged by no mental standard, and are remarkable only for their indefiniteness. The practised eye of the histologist, however, sees far more; the relative depth of cortex in various regions; the relation borne by this depth to normal standards; the varied depths of the several laminæ of the cortex, their distinctness of outline, general and local vascularity, as well as the deviations in hue dependent upon fatty or upon pigmentary changes. The œdematous, degenerated aspect of the medullary tracts, the presence of minute sclerosed patches, and a host of other morbid appearances, present to his mind a picture which the

unpractised eye wholly fails to appreciate. To those who are liable to err in the direction pointed out, I now address myself in the hope of rendering their studies of the naked-eye appearance of healthy and diseased brain more inviting and instructive, by means of a few simple directions as to what appearances are to be sought and how they are to be looked for.

A few further statements on the object and scope of these articles may not prove amiss.

I address myself almost exclusively to the student who, up to the present time, has devoted but little of his attention to cerebral pathology or to the histology of the central nervous system ; and more especially to asylum medical officers, whose opportunities for research in this field are so numerous, yet so sadly neglected. Recognising personally the want of a concise summary of methods of examination of the nervous centres, I shall endeavour here to place at the disposal of the student the more important, essential, and trustworthy information in this department which I find scattered promiscuously throughout an extensive literature. If amongst these gleanings my remarks should appear to the advanced student burdened by too much detail, my excuse must be that the primary object is to overcome the difficulties presented by these studies to the novice, difficulties which every expert observer has at the beginning to encounter.

THE DURA MATER AND VENOUS SINUSES.

General Anatomical Considerations.—It is necessary here that the student should recall to mind certain anatomical details affecting the relationships of this membranous investment of the brain which have an important pathological significance, and in the first place note that :

a. The dura mater is a fibro-serous membrane ; the outer surface being fibrous and rough, the inner being smooth and polished by a layer of epithelial cells constituting a parietal arachnoid.

b. Cut off a small portion and float it in water. The rough pilose outer surface due to the numerous fibrous connections and vessels which unite it to the inner table of the skull

becomes hereby very apparent in contrast to the smooth inner surface.

c. Next, note that the dura mater is composed of two distinct layers, which, by their divergence, occasion the formation of the different venous sinuses; whilst the inner layer, by its duplications, forms the various intra-cranial membranous partitions, the falx cerebri and cerebelli, as well as the tentorium.

d. Lastly, observe the close anatomical relationships between the lateral sinuses and the mastoid cells, and again between the superior petrosal sinuses and the internal ear. Caries of the petrous portion of the temporal bone and of its mastoid cells is so frequent an affection, that the contiguity to them of these venous sinuses is most important as likely to occasion not only localised pachymeningitis or inflammation of the opposed dura mater, but inflammation and thrombosis of the venous sinuses, leading probably to metastatic deposits in the lung.

e. The position of the venæ Galeni, which receive the blood returning from the choroid plexuses and open into the straight sinus, is such that tumours or abscesses of the mid-lobe of the cerebellum would compress them.

Upon removal of the skull-cap the student should proceed to investigate the condition of the dura mater after the following systematic manner:

1st. Note the general aspect of the membrane covering the hemispheres.

2nd. Examine the several venous sinuses.

3rd. Observe the condition of the dura mater in those regions at the base where there exists a special proclivity to disease.

1st. *As to the general appearance of the Dura Mater at the vault.*—A glance at the superficial aspect of the dura mater may reveal the presence of inflammatory products which are frequent here. If present note their character, whether they are simple, inflammatory exudates, capable of undergoing organisation, or whether they are the results of suppurative inflammation. In the former case, observe how softened the membrane is, and how readily separable from the bone; in

the latter case the presence of pus will almost certainly lead to the detection of a carious state of the internal table and diploë of the skull, and a more or less sloughy aspect of the disintegrating membrane. The organisable blastema may be found in various stages of development, as loose areolar or thick tough fibrous tissue, or it may have formed bony plates which eventually unite with the cranial bones. Should dense fibrous bands be formed, adhesions betwixt the dura mater and bone occur, chiefly along the course of the sutures, or in localised islets over the frontal or parietal bones, or so extensively that it is impossible to remove the skull-cap without at the same time including the dura mater, as their forcible separation would entail injury to the brain.

If there be the above given indications on the surface of recent or of old inflammation, note (1) the density, (2) thickness, (3) swollen (4) appearance, and (5) colour of the membrane at these parts. Recent inflammations involve much swelling, interstitial infiltration, softening and looseness of texture, and a very red colour; whilst the effects of an old inflammation are seen in a callous fibroid induration and thickening, often with bony concretions formed in the interstices of its texture, together with the adhesion to the inner table of the skull just alluded to.

The student must be prepared to make allowances for the general increase in thickness and firmer adhesion of the dura mater, which pertains to advanced age independent of inflammatory changes. Superficial inspection will also enable him to detect attenuation of this membrane such as results from the pressure of a hypertrophied brain, from the effusion of hydrocephalus or morbid growths. Local thinness of this membrane also will be frequently found due to the pressure of pacchionian bodies which perforate the dura mater frequently, and imbed themselves in fossæ on the internal surface of the cranium.

Extravasation of blood forcing the membrane apart from the bone may be frequently found, and its origin should be carefully sought.

2nd. *Examine the larger Venous Sinuses.*—A fold of dura mater close to the longitudinal sinus at its exposed frontal extremity must now be pinched up by forceps, and the

scalpel passed through it and down into the longitudinal fissure of the brain, so as to divide the union betwixt the falx cerebri and crista galli. Introduce a curved bistoury into the anterior end of the longitudinal sinus, and carry the blade backwards to its occipital extremity so as to expose this sinus throughout its greater extent. The oblique orifices of the veins of the vertex which empty themselves into this sinus will now be exposed. Note—

(1.) The capacity of the sinus throughout.

(2.) The nature of its contents.

(3.) The condition of its lining membrane.

The incision through the frontal end of the falx cerebri should now be extended on either side through the dura mater backwards, upon a level with the sawn edge of the cranial bones. The anterior extremity of the falx being then seized with forceps should be drawn forcibly backwards from between the hemispheres, carefully dividing the great superficial veins where they open into the sinus, when the convolutionary aspect of the brain covered by its soft membranes will be exposed to view. At this stage of dissection the pia mater, arachnoid, and superficial aspect of the brain will require attention, and the method to be adopted will be detailed in the following section, to which the student must be referred. Continuing, however, our examination of the sinuses of the dura mater, it must be presumed that the brain has been removed, and the following procedure will then be necessary:

Lay open the larger venous sinuses at the base, commencing at the torcular Herophili behind, and carrying the blade down each lateral sinus to the foramen jugulare. Open up the straight sinus running into the torcular Herophili in the base of the falx cerebri. Deal in like manner with the petrosal and cavernous sinuses.

A close inspection of these sinuses should be instituted with the object of learning the nature of their contents, and the morbid or healthy condition of the textures of these venous channels. The most frequent morbid conditions found here are those of phlebitis and thrombosis, the thrombus being usually the result of inflammation of the walls of the sinus.

Thrombosis of the venous sinuses of the dura mater is so

frequent and so important an affection, that the possibility of its occurrence should always suggest itself when examining this membrane.

If a thrombus obstructs any one of these sinuses, note the more or less organised condition of the clot and its adhesion to the lining membrane of the sinus.

Examine closely its apparent site of origin and extensions, the latter often reaching into the jugular veins.

Next take into consideration the constitution of the clot, whether recent, organised and firm, or breaking up and presenting the appearance of a crumbling mass, which, during life, would give rise to metastatic deposits in distant organs.

The condition of the lining membrane and tissues of the sinus should next be noted. If inflamed, the inflammatory action should be traced to its origin, usually in a carious state, of the cranial bones; or by extension of simple inflammation of the dura mater in the neighbourhood of the sinus; or less frequently, induced as a secondary result of thrombosis. The student should carefully study the effect of plugging of the different sinuses, and their proclivity to disease as a result of their varied anatomical relationships.

a. Inflammation of the longitudinal sinus will be accompanied by occlusion of the venous trunks from the convexity of the brain where they open into the sinus.

b. If the straight sinus be occluded as the result of adhesive phlebitis, the student will remark that its effects in obstructing the return of blood from the choroid plexus will be similar to what occurs when the venæ Galeni are compressed by a tumour of the mid-lobe of the cerebellum. Effusion into the ventricles might be expected in these cases, and the enlargement of the head from this cause has been recognised clinically.

c. If the lateral sinus be inflamed, we might expect as a result extension of phlebitis into the jugular veins, and plugging of the smaller sinuses or veins opening into it. In connection with this—note, that the small "emissary veins," (Santorini) which perforate the bone at the anterior extremity of the lateral sinus, and which form a communication between the sinus and the veins on the outside of the skull at the back of the head, may become plugged by a thrombus producing a

painful œdema behind the ear, also recognised clinically by Griesinger.[1]

d. The position of the petrosal and lateral sinuses naturally exposes them to phlebitis and thrombosis. Lying on the petrous portion of the temporal bone, they are always liable to be implicated by extension of inflammation from the internal ear and caries of the temporal bone.

e. Thrombosis of the cavernous sinus has been frequently noted. In connection with this condition the student must be prepared to note the pressure which will probably result to the carotid artery which runs through this sinus invested by its lining membrane.[2] He may also look for extension of inflammation of the coats of this sinus to the facial veins with which the ophthalmic vein communicates, as well as to obstruction to the return of blood from the cavity of the orbit.

f. Amongst other results of thrombosis of the cerebral veins and sinuses, will often be found punctiform hæmorrhages into the substances of the brain.

3rd. Observe the condition of the dura mater at the base where there exists a special proclivity to disease. The sites alluded to are:

1. Petrous portion of temporal bone.
2. Ethmoidal plate.
3. Parts adjoining superior cervical vertebræ.

THE ARACHNOID AND PIA MATER.

General Anatomical Considerations.—In proceeding to investigate the healthy and morbid appearances of the arachnoid the following anatomical facts will prove serviceable to the student.

1. The arachnoid is usually regarded as consisting of a parietal and visceral layer like ordinary serous sacs, the former however (parietal layer) being merely a layer of nucleated polygonal epithelium lining the inner surface of the dura mater; the latter forming the far more substantial investment

[1] Quoted by Niemeyer in 'Text-Book of Practical Medicine,' vol. ii.

[2] This condition has been recognised clinically by a loud bruit heard on auscultation of the skull. Dowse. 'Lancet.'

of the convolutions of the brain separated from them by the pia mater. This closed sac is lubricated by a portion of the general cerebro-spinal fluid.

2. The visceral layer of arachnoid does not dip into the sulci with the pia mater, but bridges them across, whilst in other regions it is widely separated from the pia mater so as to enclose between them extensive spaces. Thus at the base a delicate veil of arachnoid stretches across the pons and inter-peduncular space as far as the optic chiasma; a similar film of arachnoid closes in the fourth ventricle stretching across from the medulla to the cerebellum; the arachnoid also does not pass down to the bottom of the longitudinal fissure, but spans across immediately beneath the falx cerebri; hence a space is left here between the two inner membranes of the brain immediately above the corpus callosum.

3. The various spaces alluded to above all communicate freely with one another, and with an extensive space around the whole length of the spinal cord betwixt the pia mater and arachnoid sac. The whole system of spaces is termed the sub-arachnoid space, and is filled by the greater bulk of the cerebro-spinal fluid.

4. A space intervenes between the parietal layer of the arachnoid and the dura mater. It has been fully investigated and described by Axel Key and Retzius,[1] and is called by these anatomists the sub-dura-mater space (subduralraum). The student must therefore regard the membranous investments of the brain as consisting of a firm and immovable fibrous outer membrane, the dura mater forming also the periosteal lining of the cranial cavity; of an intermediate serous sac or arachnoid whose visceral layer moves freely with the movements of the hemisphere; and lastly, of a vascular membrane supporting the blood-supply of the brain—the pia mater, which closely lines the cortex dipping into the sulci and being prolonged into the ventricles.

Between the various investments are four great cavities:

1. The subdura-matral betwixt dura mater and the parietal arachnoid.

[1] 'Studien in der Anatomie des Nervensystems.' Axel Key und Gustav Retzius. Stockholm, 1875.

2. The arachnoid cavity formed by the arachnoid sac.

3. The sub-arachnoid cavity betwixt visceral arachnoid and pia mater.

4. The epi-cerebral space betwixt pia mater and cortex.

It must also be borne in mind that compensatory adjustments for lessened or decreased intra-cranial pressure are obtained by means of the cerebro-spinal fluid; increase in the pressure, as in the increased amount of blood, growth of tumours, &c., being allowed for by escape of this fluid into the spinal sub-arachnoid space. In relation to this important subject it has been shown by Axel Key and Retzius that the pressure of the cerebro-spinal fluid is always higher than that of the venous blood and its specific gravity lower, hence its free mingling with the blood-currents by osmosis is much facilitated.[1]

Coarse Methods of Examination.—The healthy and morbid appearance of the arachnoid should be subjected to close and critical examination.

Upon reflecting the dura mater the contents of the arachnoid sac will be revealed, and in relation to this point we must note:

1. Variations in the amount of cerebro-spinal fluid here.

2. Modified appearance of this fluid from admixture with morbid products.

3. Note expecially extravasations of blood, and the age of these hæmorrhages as revealed by the condition of the clot. It may be purely fluid blood, coagula, or a delicate film of fibrinous nature; or it may be in various stages of organisation. The various stages of the arachnoid cyst in relation to pachymeningitis have all been most graphically described.

4. Note the superficial aspect of the visceral and parietal arachnoid as regards—

a. Absence of clear, moist, glistening surface.

b. Presence of morbid deposits, such as films of lymph of varied appearance and stage of organisation; as a delicate greyish mucus-like exudation, or a membrane possessing a certain amount of consistence, or flakes which are yellow and puriform, and more

[1] 'Studien in der Anatomie des Nervensystems.' Axel Key und Gustav Retzius. 1875.

rarely fibroid patches; or, again, the development of bony plates in the parietal layer.

c. Adhesions between layers of arachnoid sac, such as occur naturally by the development of the pacchionian bodies.

5. Note the anomalies of texture due to interstitial change and to deposits occurring here. The effect of frequent hyperæmia is seen in the opalescent, creamy white or perfectly opaque appearance presented, especially along the course of the blood-vessels, and always seen after middle life to a greater or less extent. Hypertrophy of the textures, giving the membrane a tough and thick character, will result from similar causes. The membranes may be however soft, tumid, and swollen from œdema of its texture; and where it bridges the sulci a solid gelatiniform aspect is often given it by the fluid subjacent.

6. The relative amount of cerebro-spinal fluid in the subarachnoid space should next be taken into account. It should be collected, together with that which escapes from the arachnoid sac and ventricles, and carefully measured. The appearance of the arachnoid, when buoyed up by any undue amount of this fluid, should be noted before removal of the brain from the skull. It will be found that in these cases the arachnoid is widely separated from the pia mater by the subjacent fluid. The limpid character of this fluid may be tested by slitting up the arachnoid over a sulcus.

So far our remarks chiefly apply to the arachnoid, and we will now turn our attention to the pia mater. The proximity of these two membranes to one another of course predisposes to their common implication in any morbid process, yet the pia mater is occasionally involved without extension to the brain substance, and still more frequently without extension to the arachnoid. In healthy states these membranes are very thin and delicate, and removed from the brain surface only with difficulty. We should therefore note their proximity to each other over the summits of the gyri, alterations in the thickness of the pia mater and in its toughness, infiltrations of its texture, and the ease or difficulty experienced in its removal from the cortex.

Thus the pia mater may be found widely separated from the arachnoid, the latter being buoyed up by sub-arachnoid effusion. In these cases the appearance of the fluid should be noted, especially as regards the presence of inflammatory exudates, as flakes of lymph, &c.

If thickened in texture the alteration of the pia mater should be traced to its origin, whether this be simple serous infiltration or œdema conjoined with inflammatory material. Tortuosity and varicosity of the vessels will also be further indications of repeated congestions of this membrane. Especial caution must be observed against drawing hasty conclusions regarding congestion of the membranes from the mere fulness of their blood-vessels and hypostasis.[1]

The student will find ample opportunity for studying the appearances presented by œdema and thickening of the soft membranes of the brain in cases of senile atrophy, general paralysis, and alcoholism.

The presence of inflammatory material modifying the thickness and toughness of the membranes should lead to a study of the nature of the exudate, whether it appears as a tough exudate of plastic lymph more or less yellowish in hue, or more serous, sero-purulent, opaque, and less organisable material.

With the presence of these more or less plastic exudates note the general infiltration of the membranes around with greyish, opaque serum. Such changes afford excellent opportunities for the study of the results of inflammatory action in loose areolar tissue. In connection with inflammation of the pia mater, a condition which will frequently engage the attention of the student, he should also follow up his investigation of the *nature* of the exudate by examining also:

a. The *direction* taken by the morbid product, noting that this occurs almost invariably along the vascular tracts, the course of the vessels being marked out by opacities.

b. The *extension* to neighbouring structures (brain, arachnoid, dura mater, and skull).

c. The *restriction* of inflammation to *limited areas* (as when

[1] On this point see Niemeyer. Art. Hyperæmia of Brain and its Membranes. 'Text-Book of Medicine,' vol. ii.

originating secondarily from caries of the cranial bones); or to the *convexity* of the hemispheres (chiefly accompanied by plastic products); or, lastly, characterised by the *base of the brain* being originally and chiefly involved (usually accompanied by aplastic and tubercular products).

Note attentively the greater or less facility of stripping the pia mater from the cortex. The membrane may be œdematous, thickened, gelatinous, and most readily removed; or, on the other hand, it may cling with greater tenacity or adhere so firmly that portions of the cortex tear away with it, leaving an eroded, worm-eaten aspect of the surface of the gyri. Attention should be directed to the strength of these adhesions, their implication of the summits of the gyri only, their localisation over the convolutionary surface of the brain, and the coarseness of the blood-vessels entering the cortex at the sites of adhesion. Whenever the student is engaged with a case of meningitis, the morbid topography must be carefully studied. If basic, the veil of arachnoid extending from the optic chiasm to the pons must be examined and removed, noting implication of the numerous vascular and fibrous twigs extending betwixt it and the subjacent pia: follow these results of inflammatory action up along the vessels of the fissure of Sylvius and along the anterior cerebrals in the longitudinal fissure. Examine the regions just named for tubercular granulations, paying especial attention to the smaller blood-vessels which may have tubercular masses in their sheaths, occluding more or less the calibre of the vessel. Should granulations appear around the blood-vessels, let them be removed and floated in water for more careful observation.

ARTERIAL SYSTEM OF THE BRAIN.

After carefully noting the external appearance of the membranes covering the hemispheres both at the vertex and the base, the condition of the arterial system should be inquired into. The brain being placed with its base uppermost, the fine expanse of arachnoid which bridges across the inter-peduncular space is first removed, and the great vessels forming the circle

of Willis will then be exposed to view. Now separate the temporo-sphenoidal lobe from the adjacent frontal and parietal lobes by dividing the bridge of arachnoid extending between them across the Sylvian fissure. On gently drawing back the temporo-sphenoidal lobe the Sylvian branch of the middle cerebral artery will be observed running deeply in the fissure towards the upper extremity of the latter, giving off small branches in this course. The radiating gyri of the island of Reil (central lobe) will in most cases be only partially exposed, and it will be necessary to separate the operculum, or that portion of the third or inferior frontal convolution which laps over the anterior portion of this lobe. The five or seven gyri of the island will now be seen radiating outwards like a fan and supporting the various branches derived from the middle cerebral artery in the sulci between them. Split up the arachnoid which bridges across the longitudinal fissure anteriorly from one frontal lobe to the other. We shall thus have exposed the various branches of the circle of Willis as far as they can be seen at the base. Note first the arrangement of the blood-vessels.

A first glance at the arrangement of the great arteries at the base of the brain entering into the formation of the circle of Willis cannot fail to impress upon the student's mind this important fact: there are here *two great arterial systems more or less distinct and independent*. In front lies the *carotid system*, supplying by far the greater bulk of the brain; behind lies the *vertebral system*, distributed to the posterior and inferior regions of the cerebrum—an area small in comparison to the former. These two great arterial systems are united by the two posterior communicating arteries which connect each carotid with its corresponding posterior cerebral artery. Examine the posterior communicating arteries and note the remarkable smallness of their calibre, a fact which suffices to ensure us that the circulation in the carotid and vertebral systems is in the main distinct; whilst the arrangement allows of compensatory enlargement and a free communication betwixt both systems where emboli, thrombi, or diseased textures obstruct the circulation of a main branch.

Next observe the junction between the two anterior cerebral arteries deep in the longitudinal fissure by means of the cross

branch, the anterior communicating artery. Raise the branch on the forceps and note its short length, small calibre, and right-angled direction—facts which teach the student that, although in case of obstruction on the carotid side of the anterior cerebral artery, this branch by dilating may afford a satisfactory re-establishment of circulation over the area to which it is distributed, yet for the greater part the circulation betwixt the two anterior cerebral arteries is distinct.

A little further consideration will teach him that the circulation of each hemisphere is by the above mechanism rendered wholly distinct and independent; that the circulation of the mid-cerebral region of one side is wholly separated from that of the opposite hemisphere; whilst the anterior cerebral areas are more closely associated through the medium of an anterior communicating branch.

Lastly, through the medium of the posterior communicating artery (especially when we recall to mind the frequent enlargement of this vessel met with) there will be a far more ready communication betwixt the carotid and vertebral circulation than there can be betwixt the vascular apparatus of both hemispheres. The student should pay especial attention, therefore, to the following observations:

a. The almost complete independence of hemispheric circulation.

b. The very complete independence established betwixt the circulation of one middle cerebral and that of the other.

c. The interdependence possible betwixt the two anterior cerebral streams through the medium of an enlarged communicating branch.

d. The possible admixture of the carotid and vertebral circulation of the same side through the medium of the posterior communicating—a condition very frequently established.

The arrangement of the internal carotid ere it reaches the circle of Willis is one of interest and significance. Within its bony canal the tortuous sigmoid course taken by it is undoubtedly one means whereby the brain is protected from the results of the cardiac pulsations. The student will recall a

similar tortuous course taken by the vertebral artery ere it enters the cranium. It has long been noticed that the middle cerebral is more readily blocked by emboli than the other branches, and that the area of its distribution shows especial proclivity to hæmorrhage. Anatomical reasons, and the fact that this vessel lies "more directly in the way of strain from the heart explains its frequent plugging and rupture from disease." (Hughlings-Jackson.[1]) Thus Prevost and Cotard found that tobacco-seeds injected into a dog's carotid most often lodged in the middle cerebral artery. Again, the fact that the left carotid artery arises directly from the summit of the aortic arch whilst the right arising from the innominate is inclined at an angle to the aortic current, is sufficient to explain the greater immunity from embolism experienced by the right carotid in its distribution to the brain. The right vertebral arises from the horizontal part of the subclavian, and is therefore also less subject to embolism than the left vertebral which arises from the summit of the ascending portion of the left subclavian. (Duret.)

SUPERFICIAL VIEW OF ARTERIES AT THE BASE.

Note the following points:

a. Colour and opacity.

b. Relative toughness of coats.

c. Tortuosity or kinks in the course of the vessel.

d. Local bulgings or constrictions from diseases of texture.

a. The arteries may be perfectly white in cases of slow and lingering death, and contain a decolorised clot extending into their minute ramifications. On the other hand, they may be of a deep red hue from fluid blood, dark clots, and blood staining of the lining membrane. The vessels again may be thin and semi-transparent, or opaque from thickening of the arterial tunics and morbid deposits.

b. The toughness of the arterial tunics can be tested later on after their removal. Their resistance to strain is an important feature, and due attention should be paid to it by the student. The apparent thinness of a vessel should never

[1] 'Lancet,' Sept. 4, 1875.

be allowed to deceive him, as the thicker vessels are often most degenerated and least resistant to traction or expansion. Traction may be applied to the vessel held between two pairs of forceps, when an approximate idea of the breaking strain may be acquired, whilst the use of the conical gauge will enable him to note the various degrees of resistance these textures offer to expanding forces.

c. Tortuosity and kinks in the smaller branches should be noted as suggestive of previous forcible distension from congestion. This is apparent often in the larger arteries; thus Quain has noticed frequent tortuosity of the internal carotid before it enters the carotid canal outside the skull in apoplectic subjects.

d. Local bulgings may be due to aneurismal dilatations, to the different forms of arteritis—atheroma, tubercle, syphilitic gummata, or to the impaction of a thrombus or embolus. The arteries at the base will be found often exceedingly atheromatous, tortuous, knotty, and white; the amount of atheromatous material occluding even the larger cerebral arteries, often converting the smaller branches into irregular knotty cords. The student should never infer from the aspect or feel of such a vessel that it is occluded, but he should always make a section across the mass of diseased tissue, when if the vessel be permeable the orifice is readily detected. Note particularly the branches which are occluded. If the bulging be due to inflammatory swelling and exudation into the outer tunics of the artery, it will be observed that corresponding to the site of lesion the vessel is dilated—a condition due to paralysis of the muscular coat of the vessel, together with implication of the elastic outer tunic. Make a section across the inflamed tissue, and observe how readily the elastic or outer coat may be stripped from the muscular by means of forceps; also how friable and easily separable are the muscular fibres of the media. The inner coat will also be probably deeply stained by hæmatin, or may be eroded and covered by an adherent clot immediately beneath the inflamed patch. When a clot appears to obstruct the calibre of a blood-vessel, the character of the clot should be taken into consideration, its fibrinous constitution, stage of organisation, adhesion to vascular walls

its form, prolongations, and appearance of its section. The sheath and outer tunic of the arteries should be closely examined, especially in the neighbourhood of the Sylvian fissure and island of Reil for tubercular or syphilitic growths. Especial care must be taken to exactly note the arterial tunics involved, so as to discriminate between ordinary endarteritis resulting in atheromatous degeneration and the syphilitic node or gumma involving the outer coats, and the tubercular nodules involving the arterial sheaths, all of which lesions may give rise to blocking, contraction of the cavity, and thrombosis. Such lesions should be examined microscopically. In all cases the above superficial examination should be supplemented by removal of short lengths of any diseased vessels, to be reserved for freezing and section-cutting, according to the methods detailed hereafter in the microscopic section.

The vessels spreading over the island of Reil and up the Sylvian fissure should now be raised by passing a pair of forceps beneath them, and with a clean sweep of the scalpel they should be all divided just where they turn over on to the superficial aspect of the brain; this should be repeated on the opposite side. The two anterior cerebral arteries should now be divided at the genu of the corpus callosum, and dissected back to their origin from the carotids. The posterior cerebral arteries will be found running backwards round the crura cerebri under cover of a bridge of arachnoid. Together with the three cerebellar branches, they may be followed to a short distance and then divided. It will now be found that the circle of Willis is retained merely by the numerous minute nutritive branches, between the basilar and pons, and those from the anterior, middle, and posterior cerebrals, which pass through the anterior and posterior perforated spaces. Gently draw these vessels out by means of forceps as far as possible, sever them close to the surface of the brain, and then float off the vessels of the base into a shallow dish or plate of water, arranging the branches in their relative positions.

Capacity of the Arteries.—It may be found advisable in certain cases to test the relative capacity of the larger vessels at the base, and this may be obtained approximately by means of a small graduated conical gauge.

In making these comparisons the student should bear in mind that the united areas of the branches equal very nearly the area of the trunk from which they originated, although their united diameters far exceed that of the latter. It has been shown by Paget's measurements that the equality between the area of the trunk and of its branches is not exactly maintained, the area sometimes increasing in the vessels of the upper extremities and head and neck, and diminishing in the lower extremities. Now, according to the mathematical law that the areas of circles are as the squares of their diameters, it will be seen to be necessary to contrast in our measurements the square of the diameter of the trunk with the sum of the squares of that of the branches arising from it. The student therefore should proceed in the following manner: Let us suppose he wishes to estimate the relative capacity of the vessels at the base and their primary branches; let him remove these vessels as already recommended, and float them out in a shallow vessel containing water. With a pair of sharp scissors cut across the vessel exactly at right angles to its direction, pass the graduated cone into it, and gently draw the vessel on until it is fully distended, but not stretched, by the gauge; read off the diameter as shown on the gauge, and proceed in like manner with the other vessels. Arrange the diameter and the squares in appropriate columns, opposite the names of the vessels, for future comparison. The following tables represent the average measurements of the various vessels at the base in forty-five cases of insanity I have investigated:

	Diameter.	Square.
	mm.	mm.
Vertebral Artery—Right . . .	3·147	10·341
Left . . .	3·42	12·039
Basilar	3·82	14·805
Posterior Cerebral—Right . .	2·658	7·213
Left . . .	2·56	6·551
Carotid—Right	3·951	15·789
Left	4·02	16·026
Mid-Cerebral—Right	3·133	10·063
Left	3·55	10·351
Anterior Cerebral—Right . .	2·73	7·55
Left . . .	2·66	7·445

Thus the sums of the square of the diameters of the anterior

and middle cerebral of the right side was to that of the left side as 17·613 to 17·796.

The square of the diameter of right and left anterior and middle cerebrals amounted in the aggregate to 35·409, both posterior cerebrals reaching only 13·764.

Contrasting again the mid-cerebral supply of both sides with that of both anterior cerebrals, we find it represented by 20·414 against 14·995.

Reference to the table of measurements will show that the posterior and anterior cerebral supply of both sides is almost exactly similar: thus, on the right side, we find 7·213 contrasted with 7·55; and on the left side, 6·551 with 7·445.

Another interesting feature in connection with these measurements is the great preponderance in areas of the two vertebrals over the basilar, which they form by their union. The two former have as the sum of the square of their diameters 22·38, the basilar only averaging 14·805; and in several cases I have found the basilar, not more than one-half the area of the united vertebrals.

It may assist the student to obtain a clearer idea of these areas if he represents them graphically as straight lines upon an enlarged scale.

Vessels of the Pia Mater.—First note the general appearance and distribution of the vessels of the pia mater as they lie *in situ.* Observe the relative position taken by the veins and arteries, the former being large and superficial, the latter much smaller and concealed chiefly within the folds of pia mater, which dip into the sulci and are bridged across by arachnoid. The upper aspect in health being arachnoid, should present a smooth endothelial surface. Use a hand-lens in examining these vessels. The student should observe any fibrinous effusions, purulent exudates, or minute extravasations along the course of the blood-vessels, the presence of atheroma, morbid growths—as gummata, tubercle, &c. If tuberculosis be suspected, examine the arteries deep in the sulci, and this with care, as tubercle along the course of the vessels is found with far greater difficulty here than at the base or up the Sylvian fissure.

Next strip off a large portion of arachnoid and pia mater from the surface carefully, and transfer it to a deep vessel con-

taining water, the arachnoid surface being uppermost. Observe the velvety aspect of the lower surface, due to a fine pile of blood-vessels, and the very rich fringe of vessels along the intergyral folds, for the supply of the cortex deep within the sulci.

Seize with forceps one of these richly fringed folds and snip off a portion with a pair of scissors, floating it on to a glass slide, with its vessels disentangled and spread out. Remove also to another glass slide a portion of the membranes detached from the summit of a convolution, and study it by means of a hand-lens, both in reflected and transmitted light. Educate the eye in this manner into recognising the various healthy and morbid structures to be found here.

For microscopic purposes, both these slides last mentioned should be reserved, whilst a small portion of brain covered by its membrane, and including two adjacent convolutions, should also be set aside. These will afford subjects for a bird's-eye view of the surface under low powers of the microscope, such an examination being always insisted upon as especially valuable. The portion of brain must then be frozen and cut in such a direction that the sections so obtained represent both gyri with the intervening sulcus bridged over by arachnoid. We thus obtain very beautiful slides for fresh examination, which afford us the opportunity of minute examination of the cortex and the connections and relationships between it and its membranes.

The large superficial veins at the vertex where they open into the longitudinal sinus, are occasionally found plugged by thrombi; they should therefore always be examined with the object of ascertaining the nature of their contents, especially if there is evidence of inflammation of the dura mater and its sinuses, or of purulent meningitis.

The student should also take every opportunity of following up the arterial branches given off from the anterior, middle, and posterior cerebral arteries, in their course along the sulci, until he has familiarised himself with the exact supply of various regions and convolutions of the brain. It is only necessary to allude to the work of Duret and Charcot in this direction to indicate the importance of a minute knowledge of the arterial

districts of the encephalon in order to fully appreciate numerous lesions met with in the brain.

Nutrient Vessels at the base.—We should devote attention to the numerous vascular tufts which arise from the large vessels at the base for the supply of the central ganglia and pons Varolii. Those which supply the basal ganglia are disposed into anterior and posterior groups—the former arising from the anterior cerebral, anterior communicating and middle cerebral arteries; the latter taking origin from the bifurcation of the basilar, often from an enlarged posterior communicating, a further series arising from each posterior cerebral artery as it winds around the crura cerebri. Roughly, it may be stated that the anterior group supplies the corpus striatum and anterior extremity of the thalamus opticus, whilst the posterior group is distributed chiefly to the thalamus.

Note, first, their remarkably fine calibre considering their direct origin from main arterial trunks; and secondly, observe that they are given off from these trunks at a right angle—a significant fact, as they can only be affected to a minimum degree by the propulsive wave of blood caused by each cardiac systole. They are filled therefore by a sustained lateral pressure.

An examination of the nutrient supply to the pons will impress the student with the following facts:

1st. The nutrient twigs arising from the main artery are remarkably small, as in the other cases.

2nd. They also arise at right angles from the basilar artery.

3rd. The basilar is but slightly over half the capacity of both vertebral arteries which supply it.

The latter fact, which I have already demonstrated by measurement in our remarks upon arterial capacity, when taken in conjunction with the other two data warrants us in assuming that these nutrient branches are kept constantly filled by high lateral pressure and exhibit none of the phenomena of the pulse. I need not insist upon the beauty of this arrangement of means to an end. In removing the vessels at the base, be careful not to tear these nutrient vessels short, but with gentle traction draw them out and cut them across with fine curved

scissors; float the circle of Willis and its branches in a shallow dish of water, and examine the various nutrient groups. Snip off some of the anterior and posterior tufts, float them on to a slide, arranging the branches, and examine by the naked eye as well as by microscopic aid. Wherever arterial degeneration is suspected, never miss the opportunity of thus closely inspecting the nutrient supply of the corpora striata, optic thalami, and pons Varolii. The naked eye will suffice to reveal to us aneurismal dilatations along these branches, atheromatous degeneration, thrombi or embolic plugging, often explanatory of hæmorrhages within the structure of the basal ganglia or pons.

(*To be continued.*)

NERVOUS DISEASES IN VICTORIA.

BY JAMES JAMIESON, M.D., MELBOURNE.

THE question of the influence of climate in the causation of disease is a very difficult one; for this among other reasons, that it is hardly possible to eliminate with certainty social and other associated conditions. I am not prepared to say therefore that, in this contribution to the very obscure subject of the etiology of nervous diseases, much if anything has been done to make that influence plain. In itself, however, the subject is so important, and the data accumulated are so scanty, that the following statistics may be found to possess a certain value. The prevalence and fatality of nervous diseases in England have been discussed with great fulness by Dr. Althaus, in a series of papers in the 'Medical Times and Gazette' for 1876, afterwards reprinted with additions. I propose to give a somewhat similar, if less elaborate, discussion of the same points as regards the colony of Victoria. Before any comparison of the two countries is made with reference to the mortality from these diseases, certain things must be borne in mind by way of caution. Even now the population of Victoria has not the same composition as that of England as respects the proportion of the sexes and of persons living at different ages, and the difference becomes more marked as we go back to the time, nearly thirty years ago, when the discovery of gold brought so many immigrants to its shores. So great has been the change that it is not even easy to compare properly our own condition now with what it was twenty years ago, as tables subjoined will show. Social conditions too have altered greatly, and this could scarcely be proved in a clearer or more striking way than by the following fact. In

1854 there were no fewer than 70 deaths from delirium tremens, with a population of 312,000, while in 1878 there were only 16 among 879,000 persons, that number being also the average for five years preceding. The excitement and recklessness of the period, when the gold fever was at its height, subsided gradually, and must have had many other effects, though these may not be so easily established as those following the excessive use of alcohol. To these points, and to others which may suggest themselves, such as the mixed character of our population, gathered from all the ends of the earth, due weight must be given if any comparison is to be made of the results here given with those of Dr. Althaus.

I propose to compare the mortality in the two decennial periods, 1859–68 and 1869–78, thus bringing down our knowledge to the latest point, and going back as far as seems advisable in the peculiar circumstances. As preliminary to the others, the following tables supply some needful information about the population, taking the census years 1861 and 1871 as the basis. It would have been better if these could have been made the middle years of periods compared, but I have thought it advisable to give exact figures rather than mere estimates, which alone can be given for other years, and they at least afford an idea of the difference of the two periods.

TABLE I.

SHOWING THE NUMBERS OF PERSONS LIVING AT DIFFERENT AGES IN VICTORIA IN 1861 AND 1871.

	1861.	1871.
Under 5 years . .	91,514	116,688
5 to 10 „ . .	53,265	106,503
10 to 20 „ . .	67,652	140,131
20 to 45 „ . .	272,844	275,900
Above 45 „ . .	45,047	92,296
Total at all ages .	540,322	731,528

TABLE II.

SHOWING THE PERCENTAGE OF PERSONS LIVING AT DIFFERENT AGES IN VICTORIA IN 1861 AND 1871, AND IN ENGLAND IN 1871.

	VICTORIA.		ENGLAND.
	1861.	1871.	1871.
Under 5 years . . .	16·93 p. cent.	15·95 p. cent.	13·52 p. cent.
5 to 10 „ . . .	9·85 „	14·56 „	11·91 „
10 to 20 „ . . .	12·52 „	19·15 „	20·27 „
20 to 45 „ . . .	50·49 „	37·71 „	34·85 „
Above 45 „ . . .	8·33 „	12·61 „	19·43 "

Considering the smallness of the figures to be handled, I have not thought it necessary to subdivide more, and the distinction of infants, children, youths, adults, and old people, is sufficient. A gradual approximation to the English proportions is shown, but it is probable that even in 1878 differences were still very considerable. In the next table I have subdivided even less, both for the above reason and because up to twenty years of age males and females are about equal in numbers.

TABLE III.

SHOWING THE NUMBERS OF MALES AND FEMALES, AT DIFFERENT AGES IN VICTORIA IN 1861 AND 1871.

	1861.		1871.	
	Males.	Females.	Males.	Females.
Under 20 years .	106,874	105,640	186,507	185,932
20 to 45 „	190,603	92,185	169,159	114,435
Above 45 „	31,174	13,846	65,384	30,111
Totals at all ages	328,651	211,671	401,050	330,478
P. cent. of each sex	68·25 %	31·75 %	54·82 %	45·18 %

Even now males considerably exceed in number the females in Victoria, though, as is shown above, there has been a somewhat rapid approach to equality in the ten years.

TABLE IV.

Showing the Deaths from all causes, and the Comparative Mortality from Zymotic, Tubercular, Nervous, and Respiratory Diseases in Victoria in two Decennial Periods.

	1859–68.	1869–78.
Zymotic diseases . . .	34,739 = 33·06 %	34,438 = 29·56 %
Tubercular diseases . .	10,820 = 10·29 „	12,558 = 10·47 „
Nervous diseases . . .	10,751 = 10·23 „	13,196 = 11·01 „
Respiratory diseases .	8,976 = 8·54 „	12,627 = 10·53 „
All causes	105,068 —	119,848 —

Of these four classes, that of zymotic diseases showed the most marked fluctuations, as might have been expected, while that of nervous diseases did not show very great variations from year to year. To some extent there was observable a tendency to the variations in the two classes of cases to follow a parallel course, the deaths from both being greatest in the three years 1860, 1866, and 1867, of the first decennium; and in 1875 and 1876, which were great epidemic years, the mortality from nervous diseases was also much above the average. This may have been accidental, as a similar concomitant variation does not seem to have been observed in England. While the mortality from zymotic diseases compared with the total is higher in Victoria, that from nervous diseases is distinctly lower than in England, averaging 10·64% in the twenty years 1859–78 as against 12·26%.

The great blot on these statistics is the largeness of the proportion of cases vaguely registered as "Disease," and it introduces a further difficulty, in addition to those already given, in the way of a proper detailed comparison of the mortality from particular forms of disease in Victoria and in England, where there has been more strictness shown. A few

TABLE V.

SHOWING THE DEATHS FROM THE SEVERAL DISEASES OF THE NERVOUS SYSTEM IN VICTORIA, AND THEIR PERCENTAGE OF THE ENTIRE MORTALITY FROM THE SAME CAUSES.

	1859–68.	1869–78.
Cephalitis	1694 = 15·75 %	2222 = 16·83 %
Apoplexy	1503 = 13·98 „	2593 = 19·65 „
Paralysis	664 = 6·17 „	1402 = 10·62 „
Insanity	89 = 0·82 „	65 = 0·49 „
Chorea	18 = 0·16 „	15 = 0·11 „
Epilepsy	324 = 3·01 „	562 = 4·25 „
Convulsions	3881 = 36·09 „	3514 = 26·63 „
Brain disease, &c. . .	2578 = 23·98 „	2823 = 21·39 „
Total nervous diseases .	10,751 —	13,196 —

words on the general results as regards each cause may be allowable.

I. *Cephalitis* appears to be a much more frequent cause of death here than in England, where the average for a long period has been only 6·64 per cent., with a slight but steady rise of late years. The difference between that figure and the Victorian average of 16·29 per cent. is very great, and is not to be accounted for by the different constitution of the population as respects age.

II. *Apoplexy* had become considerably more fatal in the second period, probably owing in great part to the greater proportion of persons above middle life, but it is curious to note that the percentage had become higher here than in England (16·19% average, maximum 17·85%), even though the number of old people was much smaller in Victoria, as shown in Table II.

III. *Paralysis*, for the same reason doubtless as in the case of apoplexy, caused a larger number of deaths in the second decennial period, differing from it in this important respect

that even in the second the percentage of mortality was still very much lower than in England, where it had been showing a rapid and steady increase (average 15·96%, maximum 18·11%). It is not easy to say with certainty, whether the higher rate in England is satisfactorily accounted for by the greater number of old people, or whether the tendency of disease to assume a more acute course, rather than to lead to chronic degenerative changes, in the dry stimulating climate of Australia, helps to bring about an apparently slighter liability to paralysis and other forms of disease usually dependent on structural degenerations.

IV. *Insanity* has been a less frequent cause of death in the last decennium, having fallen from 0·82% to 0·49%, or only about half of the English average. The figures for Victoria are so small, however, that, independently of other sources of difficulty, it is not easy to be sure that our average is a fair one. It at least does not show any tendency to an increase of mortality from that cause here.

V. *Chorea* here as elsewhere rarely proves fatal, and the mortality of the last ten years has been just about the same proportion as in England.

VI. *Epilepsy* has shown a very decided increase, and in the last ten years had become slightly more fatal than in England, where it has also produced a steadily higher death-rate. The difference between the two countries, however, is too slight to call for observation.

VII. *Convulsions*, here as in England, form the most frequent cause of death among the nervous diseases. The mortality produced by them is almost wholly among young children, and considering that these form a considerably higher proportion of the population here it might have been expected that the death-rate would also have been higher. Quite the opposite, however, has been the case, for while the average for a long period in England has been 48·70% of the total mortality, in Victoria it was 36·09% in the ten years 1859–68, and fell to 26·63% in 1869–78, or an average for the whole twenty years of 30·88%. I do not think that cases of death from convulsions could frequently have been registered under another term, not even under "Brain disease," the symptoms

being so unmistakable.[1] The only suggestion I can make in the way of explanation is the almost complete freedom from rickets of the children in this country, that disease, according to Dr. Gee, supplying the predisposition without which convulsions are either comparatively infrequent or at least seldom fatal. Though that doctrine has been accepted by Dr. Hughlings Jackson and others, it has been sometimes pushed too far, as by Dr. Hillier. My own experience is that convulsions are far from uncommon in this country, but that the exciting cause is almost invariably peripheral irritation, and in a large proportion of cases merely gastro-intestinal irritation resulting from the ingestion of unsuitable food. It may be that the relatively low mortality is owing to the absence of the rickety state of the constitution, so that recovery is easy, the exciting cause being removable, and essentially temporary in its action.

VIII. *Brain disease, &c.*—Whilst the large number of deaths registered in this vague way makes a comparison of the Victorian conditions with those in England less easy and exact, the proportions in the two decennial periods are so nearly alike that a comparison of these is not interfered with. The diminution in the second period is no doubt owing to more care in diagnosis, and perhaps to greater exactness being insisted on by the Registrar-General.

For the sake of completeness, something may be said about the influence of sex on the mortality from diseases of the nervous system.

It is unnecessary to make any extended remarks on this table (Table VI.); but it is curious to note to what a slight extent the mortality among females had increased in the second period compared with the relative increase in the female population.

[1] Though there could scarcely be any mistake about the existence of convulsions, there is some liability to error in the form of the registration certificate; and it is possible that some deaths, actually produced by convulsions, may have been put down as having their cause in "Dentition," that refuge of the man who is careless in his diagnosis. It may be said that there is the same possible fallacy in the English figures, and that a comparison therefore still holds good. Very many deaths have been registered as from "Dentition," in Victoria, no fewer than 3716 in the ten years, 1859–68. In the second period, 1869–78, the number was only 1881, so that even if strict account could be taken, the diminution in the death-rate from convulsions of late years in Victoria must be regarded as certain.

TABLE VI.

SHOWING THE NUMBER OF DEATHS AND PERCENTAGE OF MORTALITY FROM THE SEVERAL DISEASES OF THE NERVOUS SYSTEM IN MALES AND FEMALES.

FIRST PERIOD, 1859–68.

	Males.	Females.
Cephalitis.	991 = 9·21 %	703 = 6·53 %
Apoplexy	1059 = 9·85 „	444 = 4·13 „
Paralysis	439 = 4·08 „	225 = 2·09 „
Insanity	62 = 0·57 „	27 = 0·25 „
Chorea	3 = 0·02 „	15 = 0·14 „
Epilepsy	177 = 1·64 „	147 = 1·36 „
Convulsions	2072 = 19·27 „	1809 = 16·82 „
Brain diseases, &c. . .	1629 = 15·15 „	949 = 8·82 „
Total nervous diseases .	6432 = 59·83 %	4319 = 40·17 %

SECOND PERIOD, 1869–78.

	Males.	Females.
Cephalitis	1242 = 9·41 %	980 = 7·42 %
Apoplexy	1591 = 12·05 „	1002 = 7·59 „
Paralysis	887 = 6·72 „	515 = 3·90 „
Insanity	35 = 0·26 „	30 = 0·23 „
Chorea	4 = 0·03 „	11 = 0·08 „
Epilepsy	300 = 2·27 „	262 = 1·98 „
Convulsions	1898 = 14·38 „	1616 = 12·25 „
Brain diseases, &c. . .	1815 = 13·75 „	1008 = 7·64 „
Total nervous diseases.	7772 = 58·90 %	5424 = 41·10 %

The greater number of males above forty-five years counterbalanced the effect produced by the higher proportion of

females between twenty and forty-five years. The observation of Dr. Althaus, that males are more liable to die of diseases of the nervous system than females, is confirmed by the Victorian figures; for while 58·41 per cent. of the deaths from all causes occurred among females in 1859–68, the male mortality from diseases of the nervous system was 59·83 per cent.; and in 1869–78 the proportions were 57·44 and 58·90 per cent.

No account has been taken in the above tables of the deaths from tetanus and from delirium tremens. Tetanus finds no mention in our returns of Vital Statistics, and delirium tremens is classed with the zymotic diseases, though it might perhaps with some propriety have been ranked among those of the nervous system. Judging by the mortality it must once have been excessively prevalent in Victoria, as shown by figures given at the beginning of this article. The death-rate from it has, however, undergone a steady diminution, though it is still considerably higher than that of England.

TABLE VII.

Showing the Number of Deaths from Delirium Tremens, and its percentage of Mortality among all Diseases of the Nervous System in Males and Females, and for both sexes.

	Males.	Females.	Both Sexes.
1859–68 . . .	269 = 2·43 %	45 = 0·40 %	314 = 2·83 %
1869–78 . . .	153 = 1·14 „	40 = 0·30 „	193 = 1·44 „

These figures show a distinct and satisfactory improvement, though the lower averages are still above the English, which for 1867–71 were, males 0·55%, females 0·10%. In conclusion, I append a list of the various diseases arranged in the order of their fatality, and I have thought it best to take the results of the second period, when our population had got into something like a settled condition. If the total of the percentages is found to amount to more than one hundred, the explanation is to be got in the fact that the delirium tremens figures have been added after the others were calculated.

Convulsions	26·63	per cent.
Brain diseases, &c.	21·39	„ „
Apoplexy	19·65	„ „
Cephalitis	16·83	„ „
Paralysis	10·62	„ „
Epilepsy	4·25	„ „
Delirium tremens	1·44	„ „
Insanity	0·49	„ „
Chorea	0·11	„ „

This arrangement differs greatly from that given by Dr. Althaus for England, and I am sorry that it has not been possible to give a more definite account of the causes of the differences. I have endeavoured to apply tolerably complete data, and a little consideration will enable any one interested to see how great are the difficulties in the way of any such explanation.

A PLEA FOR THE MINUTE STUDY OF MANIA.[1]

BY J. CRICHTON-BROWNE, M.D. EDIN., LL.D., F.R.S.E.

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ANY ONE accustomed to the literature of general medicine, and turning to that of psychological medicine for the first time, must, I think, be struck by the comparative paucity of reports of cases in the latter, or at least of reports of cases intended to illustrate diagnosis, or the grouping of symptoms in mental diseases. True, in our special journals, and also in non-special journals, cases of insanity may, from time to time, be found described; but these are almost invariably published, because they illustrate the success or futility of some kind of treatment, or the morbid anatomy of some coarse lesion in the nerve-centres, and not because they are typical of any variety of psychical derangement. The records of cases indeed which are issued from lunatic asylums, whatever be the motive of their publication, are singularly deficient in information bearing on the modifications of the higher cerebral functions, and many of us would be puzzled at this moment, were we asked by any zealous student, to refer him to a series of clinical delineations which would convey to him a just conception of the nature and procession of the impairments of the senses and intellect, or perversions of the emotions and will, which correspond with the commonest forms of insanity. Perhaps the best and most vivid sketches we possess are those contained in a few of our text-books, and in the writings of some of the older authors on our subject, but even these have not always been taken from nature. Some of them commend themselves to us by their literary rather than their scientific

[1] Read in the Psychological Section of the British Medical Association Meeting at Cambridge, August, 1880.

merits, sacrificing, as they occasionally do, the angularity of a fact to the rounding of a period, and darkening knowledge by mere rhetorical flashes.

If we inquire the reason of this paucity of clinical reports of cases of insanity, we shall, I believe, find that it is to be attributed to several causes. In the first place, asylum medical officers have been called on hitherto to give so large a proportion of their time and energy to administrative, sanitary and routine duties, that they have had little left to expend on minute and tedious observations at the bedside or in the wards. Without clinical clerks, or with a very inadequate number, they have had to keep their own medical records, and have therefore confined these within narrow limits, and made them of an eminently practical character. Thus it has come about that the case-books of most asylums have been kept for official rather than scientific purposes, and do not afford the information that is necessary for the preparation of clinical reports. In the second place, the medical men who enter asylum practice are ordinarily unprepared by previous training to sift and classify the special phenomena that then fall under their notice, even if they had the leisure and disposition to do so. Unacquainted with the physiology of mind, they are bewildered by its morbid manifestations, and lack that knowledge of terms and those habits of thought that are essential to an accurate analysis of psychical derangements. In the third place, the observation of aberrations of mind is exceedingly difficult and laborious, even to those who are qualified for the task, and hence few have had the intrepidity to undertake it. And in the fourth place, the attempts as yet made in this direction have not proved of much real value, while their speculative interest has not served to secure perseverance in them. No practical benefit or guidance has seemed to accrue from anything beyond a vague and general survey of the mental condition in insanity, and so we have learnt to be content with comprehensive phrases, such as excitement, depression and incoherence, and have had little encouragement to draw up inventories of symptoms, which we invariably failed to connect with pathological changes.

And yet notwithstanding this deficiency of clinical records

of insanity and disregard of its psychical aspects, most of those engaged in lunacy practice have had to give considerable attention, in certain cases, to those mental symptoms in which alone, for legal and social purposes, insanity consists. In mania they may have attempted no analysis of the overwhelming disorder of mind, but in partial insanity they have been compelled to search out its weak points, and to ascertain in what respects it has departed from its healthy state. To do this they have had to practise what I shall venture to call ideo-diagnosis. They have had to stimulate by their ideas the mind which they desired to examine. Just as in testing the electric reactions of muscles and nerves we apply the electrodes at various points of the cutaneous surface, and note the results, so in testing the state of the mind we apply our ideas, as it were, at various points of its surface; we travel over it more or less systematically, gauging its ideo-sensibility and irritability as we go, and determining as best we may, whether and to what extent they are increased, perverted, or abolished. But what we travel over in this way, when practising ideo-diagnosis, is but a scheme of mind which we have constructed for ourselves. Doubtless our skill as specialists and expertness in detecting insanity, when it is latent or concealed, will depend in great measure on the thoroughness of that scheme and the ingenuity with which we pursue the researches to which it guides us; but even the most thorough scheme hitherto devised is artificial after all, and however it may help to establish the existence of insanity, will not aid us in that medical diagnosis which we should strive to attain to. For our scheme of mind does not harmonise with any scheme of brain, and our exploration is not conducted in accordance with our knowledge of cerebral topography; and hence if we come upon what is apparently a tolerably isolated patch of diseased mind, we are utterly incompetent to indicate the whereabouts of its pathological substratum. But it seems to me that standing as we do in the light of modern researches into the functions of the brain, we cannot be content any longer with this old style of ideo-diagnosis, but must work in all cases of insanity towards a localisation of the lesion, and a recognition of its nature. We must aim at an anatomical and

a pathological diagnosis, and these are only to be reached by a careful study of symptoms and of their mode of onset and associated conditions. We are perhaps far off any success in localising and characterising mental diseases, but the endeavour to do so should at least be made, and that it may be made, trustworthy, clinical records are a first requisite; and therefore it is that I put in a plea for the minute study of mania, which will, I think, yield to minute study results of great importance.

I take it as an established fact that there is localisation of function in the brain. I cannot here enter upon the proof of that proposition, nor is it necessary that I should do so, for I dare say we are all familiar with that proof and are agreed that it is complete and conclusive. We may adopt the view that the mental operations are dependent upon separate arrears of brain-substance, or we may hold that they are connected with distinct cell and fibre mechanisms existing in a more or less diffused and interblended manner, but in either case we arrive at what is practically localisation of function in the brain; for those who hold the latter view are obliged to admit that certain portions of the hemispheres are always and principally concerned in the carrying on of intellectual and volitional operations; that the fibres and cells concerned in the conduction and elaboration of sense impressions exist in maximum quantity in different parts of the surface of the cerebrum, and that certain definite portions of the brain are in some way related to the production of movements.

I take it also as an established fact that insanity is a disease of the brain which does not always involve the whole of that organ, but which, in a large majority of cases, is localised in certain regions of it. This hypothesis is necessary to the explanation of the innumerable varieties of insanity which occur, and is borne out by pathological observations as far as they go, and by analogy drawn from the affections of other organs. It seems certain that there are system diseases and focal diseases, neural and adneural changes in the brain, just as there are in the spinal cord, and that these are severally signalised, in the brain as in the cord, by distinct sets of symptoms.

But if there is localisation of function in the brain, and if

insanity, which consists in morbid modifications of brain function, is dependent upon local lesions, we ought to be able to determine with more or less precision the position of the brain-lesions, or of its functional derangements in certain cases of insanity. The inquiry must be an eminently difficult one, for reasons which need scarcely be enumerated, but still it must be undertaken if real progress is to be achieved in psychological medicine. By the diligent study of symptoms, of the modifications of motor and sensory conduction, of reflex action, of co-ordination and nutrition, various diseases of the spinal cord have been traced to their origin, so that we can now say with confidence, in many cases, that a lesion exists in a certain white or grey tract at a certain level. Is it not to be hoped that by an even more assiduous study of symptoms in connection with morbid anatomy, we shall yet be able, in cases of insanity, to define the particular territories of the brain that are involved in the morbid process? I think this is not only to be hoped but to be anticipated, and therefore it is again that I plead for a minute study of mania, which will probably conduce in no small degree to our knowledge of localisation in disease.

But it may be objected that mania is of all mental diseases that which is least likely to afford us help in an investigation of the cerebral condition corresponding with insanity. It invades, it may be said, all mental processes, is eminently complex, and probably depends upon a disorder diffused over the whole encephalon. These objections, however, admit of satisfactory answers, and it may, I think, be shown that next to general paralysis, mania of all forms of insanity presents the most favourable opportunities for the exploration of cerebral function in disease. We are not entitled to speak dogmatically as to the nature or distribution of the pathological changes upon which mania depends, but such evidence as we possess points to the conclusion that these often partake of the character of an erythema, and are not spread over the whole of the brain, but are limited to certain regions of it. Mania is of course a comprehensive term, and includes a number and variety of mental states, from mere transitory excitement up to the wildest fury, or the most chronic madness.

And with these numerous and varied mental states diverse conditions of the brain are doubtless associated ; from a slightly heightened activity of the nervous arrangements in one or a few convolutions, with a trifling excess of the hyperæmia that accompanies ordinary functional activity, to widespread and intense capillary engorgement, or to the exudation of inflammatory products into the tissues. Fortunately we see but little of the morbid anatomy of mania, but when death does take place from exhaustion, or from some intercurrent disease during a maniacal paroxysm, the pathological appearances which we are most accustomed to find are engorgement of the vessels, hypertrophy of the brain itself, so that it expands, as it were, slightly on the opening of the skull, and vinous staining, due to capillary congestion of the grey and white matter—appearances, indeed, quite analogous to those which are seen in the skin when death has occurred during an erythematous inflammation of it. Now it is this vinous staining of the brain-substance that affords, I believe, a clue—an imperfect one, but still a clue—to the localisation of the cerebral disorder in mania, and that gives ground for the statement that even in fully developed and acute mania the whole brain is not involved. Whoever has carefully scrutinised this vinous staining will, I think, assent to the statement that it is not generally diffused, and that its most striking characteristic is, that in the medullary substance it occurs in scattered blotches. A very attentive examination of it in a number of cases has convinced me that in the cineritious substance, too, it is very unequally distributed, both in the layers of which the cineritious substance is composed and over its superficies. Although less patchy in the grey matter than in the white, this vinous staining is in the latter often confined to certain districts of the hemispheres or groups of convolution, and even occasionally to parts of particular convolutions. I have seen it strictly symmetrical in its distribution in the two hemispheres, and I have seen it much more marked in one than in the other, though never altogether confined to one. I have seen it pretty uniformly diffused over a large area of the brain, and I have seen it in distinctly isolated spots with tracts of normal brain-substance between. It is not suggested that

this vinous staining is the primary and essential change in mania, or that any connection between the symptoms of mania and the distribution of it has ever been traced out, or even between the severity of mania and its extent, and I refer to it not to argue that such a connection may be made out at present, but to indicate that the pathological conditions which subtend mania do not implicate the whole of the cortical substance of the hemispheres, a truth which I might also establish by showing that when the whole of the cortical substance is involved in inflammatory action, as is sometimes demonstrably the case in meningitis or encephalitis, a series of symptoms very different from those of mania is exhibited.

In order, however, to trace mania to its pathological seat in the cerebrum, and to render its symptoms instructive in connection with the localisation of morbid conditions, it is not requisite that we should be able to recognise its distinctive post-mortem appearances, if any such exist. To know clearly the function of a part, and to perceive in a diseased state a perversion of that function, is a sufficient justification for referring the disease to the part to which the perverted function belongs. And to know clearly the different functions of the different parts of a compound organ, and to perceive in a diseased state a perversion of certain of these functions while others remain intact, is sufficient to warrant the conclusion that the diseased state is partial in its distribution and involves only certain parts of the organ, leaving other parts exempt from attack. And so in mania, I maintain we may be justified in concluding without any assistance from post-mortem revelations, that the diseased state is limited to certain areas of the brain. During its progress certain functions of the highest nerve-centres are manifestly perverted, and these functions experiment is teaching us to regard as dependent on certain brain territories. A careful observation therefore of these perverted functions, especially when we can differentiate them clearly, will enable us to determine what brain territories are implicated by the lesions or functional derangements on which mania depends.

Now in mania the functional derangements which are presented to our observation are very numerous and of a highly

complex character. Disorders of all the mental functions, of volition, the intellect, the emotions, the propensities are displayed in a confused, inextricable, and ever-shifting mass, and these do not at present offer themselves to ready and immediate analysis. But besides these there are, I think, other functional derangements of the supreme nerve-centres, derangements of motility and sensibility which have not attracted special notice, but which will perhaps reward careful study, and afford insight into the pathology of the disease. I desire to call particular attention to the motor and sensory symptoms in mania, and to express my conviction that through a consideration of these we may attain to a greatly increased knowledge of its anatomical substrata.

The existence of motor and sensory symptoms in mania is obvious enough. Restlessness is almost indispensable to our idea of it, and every description of it that exists abounds with references to great muscular activity, contortions, gesticulations, violence, and wild cries. But these disorders of movement in mania have not been subjected to minute analysis. They have been regarded only as expressions of psychical exaltations, and as such have not been thought worthy of detailed examination. And no doubt many of the movements of maniacs are but unrestrained manifestations of ideal and emotional states, or reflexes of inordinate strength. But besides these movements there are others which, by their peculiarity and purposeless persistency, are marked out as being of a different character. And these it is which will probably, I think, be shown to depend upon excitation of the motor centres of the brain by a morbid process, and which will thus sometimes supply indications as to the parts of the brain involved in that process, and as to its lines of propagation and retrocession.

Even at the very height of acute mania, when the symptoms are infinitely complex and varied, certain markedly predominant movements may frequently be seen, which it is impossible to connect with any feeling or intention. Thus some maniacs will run about uninterruptedly night and day in a purposeless manner, and if held down in bed, will continue to move their feet and legs, as if still engaged in running. Others, again,

will remain in bed and rarely move their lower limbs, but will toss their arms about incessantly, or busy their hands unceasingly in smoothing or fraying the bedclothes. May we not suppose that, in the former class of cases, there is irritation of the postero-parietal lobule of the brain, in which Ferrier has localised the crural movements, and that in the latter class the irritation is concentrated in the ascending frontal and parietal gyri, in which the brachial and manual movements are localised. Some maniacs talk vociferously, and jargonise. May we not infer that in them there is an irritative lesion of the oro-lingual region in the third frontal convolution? Others are resolutely silent, but shake their heads from side to side without intermission. May we not suppose that in them the cortex of the superior temporo-sphenoidal gyrus is hyperæmic or inflamed?

It is not, however, in acute and fully developed mania, but in the milder attacks of the disease and in its initial stage, that useful observations of its motor symptoms may, in my judgment, be carried on. Mania may burst out at once without warning, or after a *stadium melancholicum*, in sudden completeness, but much oftener it begins in trivial deviations from customary habits of thought, feeling, and action, and attains its height by a crescendo movement. And, in the latter case, with the earliest mental wanderings come the muscular disturbances, which I regard as significant. These muscular disturbances, which may indeed precede the mental symptoms, or at least secure recognition earlier than they, are exceedingly varied in character; but I would mention, as amongst the commonest of them, fixed elevation of the eyebrows, producing a staring expressing of the countenance; habitual quivering of the upper lip, such as is seen in a child that is about to cry; twitchings of the zygomatici, rolling movements of the eyeballs, sudden clenching of the hands, and tremors of certain groups of muscles. I saw recently a case of mania in a youth in whom the malady was inaugurated by general restlessness, and a peculiar movement of the left hand. For two days before any mental disorder was apparent he brushed back his hair with his left hand every few seconds, in a rapid and abrupt manner. The movement was not habitual to him, and he

could only control it by a strong effort of will, and for a short period. Whenever his attention was withdrawn from the effort of control, the movement immediately commenced again with increased energy. In a case of recurrent mania in a lady, which I have had under observation with Dr. Ferrier, the return of the attacks of excitement is invariably announced by loud grinding of the teeth.

Now in such cases, and under such circumstances, it seems to me that the motor symptom perhaps betrays to us that region of the brain which is the starting-point of the vital irritation. Where there is grinding of the teeth at the outset of each paroxysm of recurrent mania, we might presume that the irritation, which afterwards spread over a wide zone of the cerebrum, had its centre and point of origin in or near the lower frontal gyrus. Where there is the sudden elevation and backward movement of the left hand, as in brushing the hair as a precursor of acute mania, we might believe that the upper part of the ascending frontal convolution of the left hemisphere is primarily at fault. And so where there are clenchings of the hands, twitchings of the cheeks, quiverings of the lips, rolling of the eyeballs, or elevations of the eyebrows, as preliminaries of mania, we might surmise that the ascending parietal, the ascending frontal, the superior middle frontal, or the angular gyri, were respectively the points of departure of the morbid process, or of one morbid process out of a series.

Next in importance to the observation of the earliest motor symptoms in mania, comes the observation of the order of succession of the motor symptoms as the disease advances, for this order of succession, if it could be ascertained, would disclose to us the line of march of the disease over a certain district of the cerebrum. Opportunities of making such observations in a trustworthy manner probably occur but rarely, but one such happened to me two years ago, and it was indeed this case, which I watched with Dr. Bucknill and Mr. Musgrave, that first impressed me with the extreme interest of motor symptoms of the kind which I am alluding to. A lady, aged 36, who had been depressed for a few days, suddenly became excited, restless, and talkative. For the

first few days of the excitement it was remarked that her eyes were very widely opened, and her pupils dilated. On the third day it was noticed that there were frequent sharp catching movements of the right hand, and these continued at intervals, sometimes at the rate of thirty per minute, until the fourth day, when it was remarked, the patient being then in bed, that there were constant flexion and extension movements of the right foot, or the ankle. On the fifth day, and for many days afterwards, the movements of the foot having ceased, there were incessant rhythmic waving movements of the right hand and arm. The left arm lay at rest, but the fingers of the left hand were constantly twisted, the middle finger being turned over the dorsal aspect of the ring-finger. Here it appeared warrantable to suppose that one arm of the morbid process had stretched along, during a period of three days, from the posterior end of the upper frontal convolution through the upper extremities of the ascending frontal and parietal gyri to the postero-parietal lobule on the left side, and had also run down the ascending frontal convolution, while on the right side the ascending parietal convolution was the only part of the motor area implicated.

It is not to be expected that cases of mania, with motor symptoms admitting of such a simple interpretation as has here been offered, will be frequently encountered. In most cases the motor symptoms are intricately complex, and are associated with voluntary emotional and reflex movements, in a way that defies all attempts at separation, and in few cases does the morbid process pursue a course determined purely by tissue continuity. It is probable that the irritative condition of the cortex is spread and propagated, in many instances, in accordance with the functional alliances of different parts, or with their vascular supply, and then of course it would be in vain to look for a succession of symptoms corresponding with the arrangement of centres in any region of the brain. Still it may be occasionally possible to obtain glimpses of the lines of invasion and retreat of irritative lesions through the motor areas of the brain through the order of succession of the motor symptoms, and that order of succession is therefore deserving of careful study.

But there are sensory as well as motor regions in the brain, and sensory as well as motor symptoms in mania, and a judicious investigation of these sensory symptoms will, in all likelihood, help us still further to understand the nature and distribution of the pathological conditions which underlie the maniacal state. Hallucinations of sight, hearing, taste, smell, and cutaneous sensibility, and impairments of sensory perception in connection with any of these, are difficult to identify and isolate. At the inception of a malady, however, certain retinal or Schneiderian or cutaneous projections may now and again be singled out, and then become the key to some of its secret recesses, opening up to our inspection, as it were, certain pathways of pathological inroad. As they are, however, purely psychical states, as opposed to physical symptoms, and as the statements of a maniac are rarely reliable, their existence is much more difficult to establish satisfactorily than is that of morbid motor phenomena. And even when they have been proved to exist, their interpretation is beset with difficulties. Bastian has pointed out, as I think with great justice, that the perceptive centres for visual and also for acoustic impressions, have probably a widespread seat in the cerebral hemispheres, while those pertaining to the gustatory and olfactory senses have presumably a more limited distribution. And yet, when we have in any case of mania distinct evidence of an optical illusion, or coloured vision, at the commencement of the attack, or persistently through the whole of it, I should be disposed to diagnose an irritative lesion, including incontinent discharge in the angular gyrus. And where, again, we have auditory hallucinations I should be disposed to diagnose a similar state of things in the superior temporo-sphenoidal convolution; then if in any case we found hallucinations of taste, or marked imperfection of that sense, with muscular movements of the lips, tongue, and jaws with those movements, in fact which are usually consequent on the excitation of the gustatory sensation, we might with propriety hazard the opinion that the middle temporo-sphenoidal convolution was the seat of morbid irritation. Where, again, we found hallucinations of smell, or of tactile sensibility, and these are by no means

uncommon in the early stages of mania, we should naturally refer these to lesions of the subiculum, and of the uncinate gyrus and hippocampus major.

And here it may occur to us that in our attempt to elucidate and connect the symptomatology and pathology of mania, we have overlooked the facts that there are extensive regions of the brain, the præfrontal lobes, which have yielded to experiment only negative results as regards both sensation and motion, and that there are grounds for believing that the centres for the highest mental functions are located in these lobes, and that in a large proportion of cases of insanity the essential morbid changes have their starting-point and home in them. But we are not left altogether without indications of the existence and progress of disease in it. I dare say we are all familiar with cases of mania which begin in a singular hebetude, or in a suspension sometimes sudden of voluntary and varied activity. The patient's muscular powers and sensation are unimpaired, he comprehends all that is said to him, and does not labour under any delusions or hallucinations, but he stands fixed and motionless, or performs any actions to which he may be urged in a stiff and machine-like way. His muscles are perhaps slightly rigid, and his whole aspect suggests that he is under a severe duress which checks all manifestations of spontaneity. He is, I should say, over-inhibited. But a little later a singular change has taken place in his condition. He is no longer statuesque, but restless. Hebetude has given place to animation, still without delusions or hallucinations; he is fitful and inconsiderate, being swayed inordinately by every passing impression that is made on him. He is incapable of concentrating attention upon any subject or of remaining at rest. He is now, I should say, under-inhibited. The highest controlling centres, which were in the first instance stimulated, have now become paralysed. In the first state they arrested the activity of the centres that are subordinate to them, and now their control has been withdrawn from these centres, which respond therefore in an excessive and extravagent manner to all appeals made to them. But the highest controlling centres are located in the frontal lobes, and the conclusion therefore is

that in cases of mania beginning in the manner just described, the disturbance of the cerebral cortex starts from that region. No doubt lesions in this large region of the brain will be followed by very varied assemblages of symptoms according to their position and extent, and the cases to which I have adverted illustrate only one mode of invasion of disease in this region. We shall most of us recognise, I fancy, in Ferrier's account of the effects of destruction of the antero-frontal lobes in monkeys an accurate portrayal of the first stage of mania in another and numerous class of cases. "I could perceive," he says, "a very decided alteration in the animal's character and behaviour, though it is difficult to state in precise terms the nature of the change. The animals operated on were selected on account of their intelligent character. After the operation, though they might seem to one who had not compared their present with their past, fairly up to the average of monkey intelligence, they had undergone a considerable psychical alteration. Instead of as before being actively interested in their surroundings and curiously prying into all that came within the field of their observation, they remained apathetic or dull, or dozed off to sleep, responding only to the sensations or impressions of the moment, or varying their listlessness with restless, purposeless wanderings to and fro."

My object in this paper has been merely to point out what appears to me to be a new and promising method of investigating mania, by a close observation of its symptoms in the light of recent researches into cerebral localisation. I could not within the limits assigned to it do more than indicate in a few instances the kind of symptoms that should be specially observed and the manner in which they may be translated into intimations of cerebral disease, and I have not therefore been able to make use even of the scanty stock of facts bearing on the subject which I have as yet been able to collect. If I am correct in believing that mania is immediately dependent on irritative lesions of the cerebral cortex which invariably involve the highest centres, but which very irregularly and unequally invade the sensory and motor regions of the hemispheres, then an abundance of facts will not

long be wanting to verify and illustrate the proposition and to explain away difficulties like that which presents itself when we remember that irritative lesions of those very centres which in mania we suppose when irritated to cause movements of the kind adverted to, in other conditions result in convulsions and chorea. It may be that the coincident involvement of the higher controlling centres in mania, and their exemption from participation in the irritative state in convulsions and chorea, accounts for the difference in the phenomena observed in these disorders, in all of which we assume the same motor centres to be morbidly irritated, or it may be that differences in the kind and degree of the irritative condition affords the key to the solution of the problem; but on such points we can only vaguely speculate at present, and until much careful investigation has been carried out.

But it may be demanded what advantage is to be secured by even the most detailed study of the movements, hallucinations, and thought-currents in mania? Supposing that all these had been traced to their starting-points in the brain—an altogether extravagant supposition—and that we were able to put our finger on the gyri in which the disorder originated in each case, what nearer should we be to its curative treatment, what reward should we reap for our patience and pains? It is not for me here to answer at any length such old-fashioned and short-sighted interrogatories, or to defend the acquisition of knowledge for its own sake even when that acquisition calls for protracted toil. All knowledge brings its own exceeding great reward, but not to those who pursue it in a purely mercenary spirit. But more than this; knowledge never fails to bring with it gifts that were not contemplated when it was sought. For a time it may seem to stand, solitary and useless, serving only as a monument of the ill-directed industry of him who quarried it and set it up; but in due season, as the building of the temple of science proceeds, the place is found which it and it alone can fill, and it becomes an integral and essential part of the goodly structure. If then an intimate knowledge of the phenomena of mania promised no immediate or practical return, it might still be pursued in confidence that

some unforeseen recompense would be bestowed on it. But even now some valuable fruits that may be gathered from that knowledge may be discerned. In the progress of pharmacology it has been established that certain drugs act specifically on certain centres in the spinal cord, medulla oblongata, and basal ganglia; and it is not too much to anticipate, when certain functional areas and centres have been satisfactorily differentiated in the brain, we shall soon be put in possession of means of modifying their activity and of arresting some of the disordered states into which they are liable to pass. Our experience of cannabis Indica, hyoscyamus, stramonium, belladonna, strychnia, chloral and other drugs, points to a preference and priority of action in each over definite cerebral tracts and regions, and a further experimental employment of these drugs, and of other therapeutic agents which will certainly be put into our hands, in the light of an enlarged acquaintance with cerebral localisation in health and disease, cannot fail to conduct us to conclusions of the utmost practical utility. Groping in the dusk as we do at present, we occasionally find our way in cerebral disease to the application of an appropriate and potent remedy. Walking in brilliant illumination, as we may perchance do hereafter, we should achieve such triumphs of our art, not occasionally, but habitually.

Critical Digests and Notices of Books.

Die Schrift-Grundzüge, ihrer Physiologie und Pathologie. By Dr. A. Erlenmeyer. 8vo. Stuttgart, Bonz & Co. (pp. 70, and 12 plates).

The first part of this essay is devoted to some physiological and ethnological considerations on the art of writing. We shall not follow the author into this portion of his task, but devote the space at our command to giving the reader an account of his views about the pathological changes observed in the handwriting of patients suffering from various diseases.

He divides alterations of writing into mechanical and psychical. In the former, which may be divided into ataxic and tremulous, the shape of the letters is interfered with; in the latter, included under the name of dysgrammatography, there are errors of orthography, grammar, and syntax. Dysgrammatographies fall under the headings of *conscious* (agraphy and paragraphy) and *unconscious* (writing in general paralysis of the insane).

The ataxic handwriting is characterised, like the ataxic gait, by the excessive execution of the movements necessary to the formation of single letters. Hence the up-strokes are carried on beyond their proper limits, the down-strokes are thicker and longer than necessary, the curves are angular and large, the letters of irregular size, the words do not preserve the straight line. Physiologically, this state of things obtains in the child's first attempts at writing; pathologically, it is found in cases where some central or peripheral lesion interferes with the due co-ordination of the various muscles involved in the act of writing. It is also found in cases of convalescence after grave acute disorders, such as typhus. It naturally supervenes in cases of sclerosis of the posterior cervical columns and in cerebellar diseases, and occurs "functionally" in the

condition of fatigue, and in intoxication, through alcohol and chloral.

Certain forms of writer's cramp give rise to ataxic writing, which, it must be added, does not always occur pure, but also in combination with the tremulous and paralytical types.

Tremulous handwriting is observed physiologically among old people, and consists simply of a wavy outline of the up and down strokes. In every other respect the writing is perfect. It is induced by cold, and by a variety of poisons, such as alcohol, morphia, chloral, nicotine, &c.; but as the author remarks in the case of alcohol, for instance, it is not when under its influence that the patient writes tremulously (he then writes ataxically), but when fasting. A small quantity of alcohol may even momentarily restore firmness to his muscles.

Tremulous handwriting characterises disseminated sclerosis, and also occurs as an element in many cases of general paralysis. The author is not able to say whether the difference in the characters of the tremor in the former disease and in paralysis agitans manifest themselves by their respective handwritings.

Experimental proofs of the implication of the various muscles involved in the act of writing can be readily obtained by localising an interrupted current of electricity in each of them on a healthy subject. A clear notion is thus gained of the difference observed between vertical and lateral tremor; the former is due to implication of the musculo-spiral group of muscles only.

Under the heading of psychical disturbances of handwriting, the author includes agraphia and paragraphia. Much the same considerations which arise in the case of aphasia obtain also with reference to agraphia. This may be amnesic or ataxic. But the author insists on the distinction between true paragraphia, the subject of which is conscious of his not writing correctly, and the paragraphia of the general paralytic, who shows no such consciousness, but is perfectly satisfied with the nonsense he may be writing.

The author discusses and illustrates the following propositions with reference to "paralytical" writing. It occurs only in presence of a diffuse cortical lesion. It is characterised by

excess or deficiency: necessary elements of language (letters, syllables, words). It appears very early in the disease, and may then be a valuable element in the diagnosis.

With the progress of the lesion it becomes associated with the mechanical disturbances previously considered. The author particularly insists upon the diagnostic importance of a careful examination of the handwriting in the early stage of suspected general paralysis, and shows by example how it may be of assistance in doubtful cases. Another important point is, that in those cases where a syphilitic process is at the root of the evil, and where an appropriate treatment is being successfully pursued, the first symptom of improvement is given by the restoration of correct writing. The comparison between handwritings collected at different periods of the disease is in all cases the easiest way to estimate the progress the patient is making for better or for worse.

The author gives an instance of the temporary benefit derived from galvanisation of the brain in general paralysis. Under the influence of the current there is an improvement both in the mechanical and psychical abnormities of the handwriting, and it is to be noted at the same time that the patient writes more rapidly than before. He adds, however, that he has never derived any permanent results from a systematic application of galvanism in cases of general paralysis.

Dr. Erlenmeyer illustrates his essay by means of a series of facsimiles of well-chosen examples, and deserves all credit for having led the way in the attempt of giving a systematic account of the pathology of handwriting, and opening up a fruitful field of study to the clinical observer.

A. de Watteville.

Brissaud on Hemiplegic Contracture. 'Recherches Anatomo-Pathologiques et Physiologiques sur la Contracture Permanente des Hémiplégiques, par E. Brissaud. 8vo. pp. 206. Paris, 1880.' (Publications du Progrès Médical.)

In this able and instructive monograph, highly worthy of the distinguished Salpétrière school of neuropathology, M. Brissaud

discusses the morbid anatomy, clinical features, and physiological pathology of hemiplegic *contracture.*

Hemiplegic contracture, secondary degeneration in the spinal cord, first signalised by Bouchard, and cerebral localisation, being all intimately related questions, it is necessary in the first place to consider the anatomy, normal and pathological, of the internal capsule and pyramidal strands, and the connections of the latter with the cortex on the one hand, and the spinal cord on the other.

The pyramidal strands are to be considered as connecting the motor regions of the cortex cerebri with the multipolar cells of the anterior cornua of the spinal cord. They pass through the internal capsule and cerebral peduncle, decussate more or less completely (occasionally not) at the lower anterior aspect of the medulla oblongata, and then descend in the posterior part of the lateral column, gradually diminishing in their progress until they almost disappear at the lumbar region. The non-decussating strands descend in the antero-internal column of the same side intimately related to white commissural fibres which probably connect the pyramidal strands with the lumbar region. There is no good ground for supposing with Huguenin that the pyramidal strands form a direct connection between the cortex and the muscles; but rather that they are commissural between the cortex and the motor cells of the anterior cornua.

Bouchard first indicated the relation between secondary degeneration and lesions of the internal capsule; but Charcot first clearly differentiated the effects of lesions of different parts of this structure. In 1875 the anterior two-thirds of the internal capsule were regarded as belonging to the motor region, and the posterior third to the sensory paths. But the researches of Flechsig in particular, founded specially on the developmental anatomy of the brain, have considerably circumscribed the area of the internal capsule directly connected with the motor regions. On horizontal section of the hemisphere (which displays the form of the internal capsule better than the vertical sections of Pitres) the internal capsule is seen to be formed of two segments: an anterior, directed forwards and outwards, and bounded by the caudate and lenti-

cular nuclei, internally and externally respectively; and a posterior division, directed backwards and outwards, and bounded by the optic thalamus and lenticular nucleus. The point of union of these two segments is termed by Flechsig the *knee* or *genu* of the internal capsule. From Flechsig's researches it appears that the motor regions of the cortex have no relation with the anterior segment, nor with the posterior third of the posterior segment, but only with that portion intermediate between these two. But though it would appear that the pyramidal fasciculus is confined to this portion, yet secondary degeneration may occur in the internal capsule outside this area. This is the first point on which M. Brissaud gives the results of his own investigations.

Secondary degeneration when it occurs is most evident in the inferior aspect of the cerebral peduncle. When this is carefully examined it may be differentiated into three fasciculi. These, again, are divisible as follows:—

1. A posterior or sensory fasciculus which, according to Meynert, passes into the occipital lobe. This is never the seat of secondary degeneration.

2. A middle fasciculus, which is the most common—and which has usually been considered as the sole—seat of secondary degeneration. This contains the fibres conveying volitional impulses to the trunk and limbs.

3. An internal fasciculus, which has long been regarded, but erroneously, as incapable of degeneration. This fasciculus is of small dimensions, and may be termed the "geniculate fasciculus," as it is connected especially with the "*genu*" of the internal capsule. This fasciculus contains motor fibres which are distributed to bulbar centres, and specially move the face, the tongue (perhaps also the soft palate), and in general all the parts of the head and face capable of volitional movement.

4. A fasciculus (internal to the preceding) which also terminates in the bulb, degeneration of which does not seem to be associated with other than intellectual disorders.

When these four fasciculi are examined in their relations with the different portions of the internal capsule, it appears that—

1. The posterior fasciculus corresponds to the posterior third

of the posterior segment. This is the region of hemianæsthesia.

2. The middle fasciculus corresponds to the anterior two-thirds of the posterior segment—the pyramidal fasciculus.

3. The geniculate fasciculus corresponds to the genu of the internal capsule, and is in direct connection with the third frontal convolution or inferior extremity of the ascending frontal.

4. The internal fasciculus corresponds to the whole of the anterior segment of the internal capsule.

Various cases are detailed illustrative of these generalisations, which if substantiated in all their precision by succeeding research, must be regarded as a great stride in structural and functional localisation.

In the spinal cord the degeneration affects the lateral column, the same tract as is primarily invaded in amyotrophic sclerosis. But as the pyramidal strands end in the cells of the anterior cornua, it thus becomes possible for degeneration of these strands to extend into the cornua and lead to atrophy of the cells with its consequences—muscular atrophy. That muscular atrophy may occur in connection with hemiplegic contracture has long been maintained by Charcot, and Brissaud adduces evidence both from the descriptions of older observers, such as Todd, and from recent cases, to show that muscular atrophy is much more common in connection with hemiplegic contracture than is generally supposed.

In the cases in which this has been found, there is marked diminution of the anterior cornua of the paralysed side, and the large cells greatly diminished in number, or entirely gone. Those which remain show evident signs of degeneration. The cell groups most commonly affected are those in the anterior extremity of the cornua. The gelatinous substance surrounding the cells is also altered and atrophied. The cells of the intermedio-lateral tracts are also frequently invaded by the atrophic degeneration. As in the case of the white columns, the atrophy is most pronounced in the cervical region, occupies less extent in the dorsal region, and is scarcely visible in the lumbar region.

Hemiplegics have been usually divided, according to Todd,

Duchenne, &c., into two great classes, viz., those with, and those without contracture. These latter, however, when the hemiplegia is incurable, are comparatively rare, and it is doubtful whether they ever occur without a disposition to rigidity, characterised by exaggeration of the tendon reflex and spinal trepidation. Hence Brissaud would reject the category of permanent hemiplegia without rigidity, and term it hemiplegia with *latent* contracture. Hemiplegics are frequently met with who have apparently recovered and escaped the usual contracture, and who nevertheless are incurably paralysed. Several cases are related of this order in which the limbs, otherwise flaccid, are at once excited to contracture by a tap on the tendons. They are in a state ever ready to exhibit contracture, though not permanently rigid. In the same way that spinal reflexes excite spasm, so do volitional impulses, emotion, &c., excite the same. In hemiplegia with contracture, permanent or latent, the phenomena of *associated movements* are frequently observed, i.e., a propagation from the sound to the paralysed side, and *vice versâ*. Thus in a case of right hemiplegia with latent contracture, closure of the left hand excites the same action in the right, and when repeated several times in succession gives rise to actual contracture of the right hand. In another case, left hemiplegia and slight contracture, closure of the right hand increases the contracture on the left. In this case also closure of the left hand by itself is not possible until the movement has been first initiated by the sound hand. Similarly also, as Déjerine has shown, there is often a propagation of spinal trepidation from the paralysed to the sound side. In fact, as will be seen subsequently, the condition as to reflexes in the sound side of a hemiplegic is never quite that of a normal individual.

It is not merely as regards contracture and its allied conditions that there is a passage from one side of the cord to the other. It is possible for a paralytic condition to extend across to the sound side. There are in the wards of the Salpétrière a great many hemiplegic females who after a certain number of years have become paraplegic, with rigid flexion and immobility of the limbs. These cases are no doubt to be explained by a transverse extension of the sclerosis.

Another transformation of hemiplegic contracture is a return

to the state of flaccidity. This coincides with the setting in of muscular atrophy. This may occur at a variable period, and is most marked in the shoulder, and thenar eminence. It is not to be confounded with mere wasting from disuse; when it sets in the contracture gives way, and the tendon reflex diminishes or disappears. The muscles which become atrophied are frequently the seat of acute pains, which coincide with the atrophic degeneration.

A special chapter is devoted to the description and investigation of the "signe du tendon," or what is very commonly called tendon-reflex. On the setting in of hemiplegic contracture the patellar tendon reflex becomes exaggerated. Changes also occur in the time, amplitude, duration, and form of the contraction.

Brissaud's measurement, determined by a careful method which he describes and figures, of the normal time is somewhat higher than that given by Tschiriew. Tschiriew gives 32 to 34 thousandths of a second as the interval between the percussion and contraction; whereas Brissaud puts it at from 48 to 52 thousandths. Gowers' measurements, 0·09″ — 0·15″, are not regarded as accurately made. If we take 50 thousandths of a second as the mean, we can only take it as an average, as the tendon reflex is liable to variation from numerous causes. But the chief point is that in the normal individual the two sides are alike in this respect.

The amplitude cannot be determined absolutely; the duration of the contraction is usually from one-third to one-half second; and the form of the contraction is like the ordinary muscle curve.

In hemiplegic contracture the time of the reflex on the paralysed side is diminished, usually by 4 to 5 thousandths of a second, as compared with the sound side. But it also appears that the so-called sound side is not actually altogether normal. For while the normal average is, as already stated, 48 to 52 thousandths, in the sound side of a hemiplegic the average is between 38 and 42. This fact throws light on the mechanism of associated movements and other phenomena above described.

The contraction is also more brusque, and the duration is

also greater, as is readily apparent on comparing the cases with each other. The duration of the contraction is not unfrequently double that of the normal, and the lever often shows dicrotic or polycrotic curves in its descent.

Another point in reference to the tendon reflex is that, though percussion of the ligamentum patellæ is not at all painful, yet, if frequently repeated, it gives rise in the normal individual to a peculiar sensation, difficult to describe or define, but which is felt in the lumbar, dorsal, or even cervical region.

In hemiplegic contracture this is very marked, and has almost the character of an *aura* running up the dorsal region to the throat. In fact, an epileptiform convulsion may result, as illustrated by a case under the care of M. Debove.

The state of contracture may be characterised as a state of *permanent muscular activity.* This is shown by the giving way of the contracture when the limb is rendered exsanguine by Esmarch's bandage applied for about twenty minutes, except in cases where the contracture has resulted in deformity of the articulations.

With respect to the attitude assumed by the limbs, viz., flexion with pronation of the upper extremity, and extension with adduction of the lower extremity, various hypotheses have been advanced to account for it. But experiments with strychnine show that the attitude assumed in the tetanic spasm is not due to the action on one set of muscles only, but is merely an exaggeration of the normal muscular tone; and the condition is precisely the same in contracture, the position assumed being that of equilibrium between the flexors and extensors, constituting, as Charcot terms it, a *spontaneous strychnism.*

Among theories advanced to account for contracture have been encephalitis or myelitis; removal of the inhibitory influence of the cerebrum; peripheral neuritis; associated movements, all of which are more or less inconsistent with the real facts. The contracture is most satisfactorily accounted for on the hypothesis of irritation of the cells of the anterior horns by the secondary degeneration in the pyramidal strands of the cord, an explanation which harmonises best with the other conditions in which contracture manifests itself, as in strychnism, primary lateral sclerosis, or contracture dependent

on transference of sensory irritation, as sometimes occurs in tabes.

The contracture lasts only so long as the cells of the anterior horns are intact. When they degenerate the contracture ceases, and the muscles atrophy.

Looked upon as an exaggeration of the normal tonus, contracture may be considered as a kind of permanent reflex contraction, like tetanic spasm, but less violent, and more persistent. The equilibrium which characterises the hemiplegic contractures is liable to variation under various influences. Thus emotion increases it, so also electrisation, as Duchenne has shown. They all have the effect of increasing the excitability of the spinal centres. Strychnine increases it very markedly. Among other causes also deserving special mention are traumatic injuries, especially about articulations, whereby the nerves specially calling forth muscular contractions are irritated. Hysteric contractures have essentially the same characters.

Latent contractures depend on a hyperexcitability being maintained by a process of degeneration not extensive enough to set up the state of contracture, except on further provocation.

In a similar manner we may explain the occurrence of irregular movements of the type of athetosis which are often observed in limbs not entirely paralysed. During the waking state the muscles are never allowed to be absolutely at rest. The contraction of one excites others reflexly, and so the limb is in a constant state of movement. The spasmodic tendency in these cases is mostly dependent on lesion high up, in the lenticular nucleus or of the thalamus, and in immediate contiguity to the internal capsule.

As regards the physiological condition of muscles in a state of contracture, it has already been shown by the Esmarch bandage that the state is one of activity. Yet it is not contraction, for a contractured muscle can be made to contract by stimulation. The condition is one intermediate between flaccidity and contraction. The state of repose is a state of tonicity, not of flaccidity, and therefore as tonicity does not induce muscular fatigue, we can understand how the prolonged exaggeration of this tonicity does not cause any evident

fatigue. But one would expect in the state of contracture the chemical and physical phenomena which accompany contraction, though in a less degree. Among these are two which Brissaud has specially investigated, viz., the muscular bruit, and the generation of heat. The bruit of the *contracted* muscle is regular, sonorous (*bruit rotatoire*) and constant in the number of its vibrations; that of the *contractured* muscle is feeble, irregular, and jerking, with interruptions and intermissions. The muscular fibres seem, instead of contracting regularly and simultaneously, to follow each other and to act with different degrees of strength.

The contracture ought according to theory to be accompanied by development of heat. On this point Brissaud has had the aid of Regnard in his investigations, but so far as they have gone the facts seem contrary to their expectations.

The contractured muscles of hemiplegics are in reality always of a lower temperature than those of the sound side. The difference is slight—several tenths of a degree—but constant.

They endeavour to explain this by the heat generated in the sound side through the movements requisite to place the patients under the conditions necessary for investigation, continuing some time, and thus vitiating the comparison. And they are of opinion that the comparison could only be accurately made during sleep, which is practically impossible.

But it may be suggested that the result could be arrived at notwithstanding these difficulties. It is quite possible for the contractured muscles to be of a lower temperature than those of the sound side, and yet be higher than in the state of flaccidity. Hence comparative experiments on both limbs before and after the influence of chloroform, which causes the contracture to give way, ought to afford a simple solution of the problem.

In addition to the facts and arguments contained in the body of the memoir, of which the above is a *résumé*, M. Brissaud appends the details of numerous illustrative cases. The subject of which he treats is one of the utmost importance in neuropathology, and the ability with which it is handled is worthy of the highest commendation.

DAVID FERRIER.

Scientific Transcendentalism. By D. M. London: Williams and Norgate. 1880.

The title of this work will probably be considered by many minds to be somewhat anomalous; for "Transcendentalism" is too often thought to be beyond the region of science altogether, and not to differ much from whatever is impossible or absurd; and, at any rate, it might be incongruous to attempt to apply the method of one department of knowledge to that of the other. After all, the difference is very likely only one of degree, and that party may by no means be the only one entitled to certainty, which endeavours to secure it by adhering rigidly to nature in the concrete form, and never using the intellect, save as a rake to collect what lies upon the ground. Of course there is no need here to discuss whether a science of metaphysics is possible, and to do so would involve opening the aged question of Idealism *v.* Materialism. But even ultra-materialists may be induced to abate their scorn by the sight of the precision—even mathematical—with which our author gives sensible proof of his intentions, unless they suspiciously regard it as a snare laid by the Evil One to entrap the truthfully pure: moreover, the volume is so small (pp. 113), that it cannot repel the most dainty appetite on that score. But, lest readers should fancy that the sole attraction of the book consists in the establishing of a system of Ontology, we desire to call attention to another feature, which might not be looked for, as it is not so very often to be met with in works of this class, and which makes this one a valuable object of study—even in medicine as well as psychology—as well independently, as from its bearing on the special contents of the work; we refer to the account given in the introduction of the singular genesis of the doctrines here set forth, and which makes the work an important contribution to the psychology of mysticism; and if readers care not much for the picture these doctrines form, they will assuredly do so for the canvas on which this picture is painted.

This preliminary chapter occupies a considerable portion—nearly one-fourth—of the whole; it commences, as might be expected from a professed Transcendentalist, with a reproving

criticism of the claims of modern science, on the ground that, though a large accumulation has been made of late of material facts and inductions therefrom, and therefore "careless persons are in danger of believing that nothing more, or not much more, remains to be discovered;" yet nothing whatever has been gained in the department of mind. But there are always some, and our author is one, who are too deeply convinced that a science higher than physics and chemistry really exists, and that the method of intuition is to be relied on. Now, if it can be shown that the canons of ordinary science are equally applicable to the superior field, the certainty allowed to the former may be extended to the latter; this D. M. claims to lay forth. In order to be in possession of a universal theory, he will not attach himself to the position of common Transcendentalists, who believe in the dependence of phenomena on a Person of Infinite Power outside the universe subjecting it to moral guidance; but fully acknowledges the objections of materialists that this fails to account for the laws of matter, however well it might explain those of mind. Thus, the Materialist and Transcendentalist theories both contain much, but not the whole truth: will not the adoption of the plan of theorising in common experience lead us up to the greatest theory? Now, this process consists in seeing a resemblance between a new unknown and that which is already known; the Transcendentalist, therefore, has to assimilate matter to mind, the Materialist mind to matter. Here, then, lies the problem of philosophy, to resolve the two into unity.

So far we meet with nothing extraordinary; however improbable a fantasy such doctrines seem, they are at least to be found in cool prose from hundreds of other sources. But may we not be assured that the tones proceed from Bedlam, if not from the sepulchre, as he unfolds, on pp. 6–7, the amazing tale of his escape from the prison-house of sense? We may advantageously quote a paragraph or two in full:—

"Four years ago nothing was farther from the mind of him who writes these lines than the desire of becoming an author of books, a man of science, or a public theorist on any subject whatever. He was then in the service of a banking company in the interior of South America. He had a taste for modern

languages and comparative philology; read the Bible, Plato, Shakespeare, and Jeremy Taylor; taught the Gospel of Jesus Christ in the Sunday School; and tried to do his duty, as far as he knew it, in the least offensive way. He had acquired the, in that country, contemptible character of 'hombre sin vicios,' which it was part of his creed to maintain.

"One morning, however, this quite commonplace individual awoke to find himself possessed of a curious power of inventing allegories or parables on all sorts of moral and metaphysical subjects. Conceptions chased each other through his imagination like dissolving views. Occasionally a brighter image than the rest would stay long enough to be described: it would float before the eyes like a fairy scene in a pantomime, blotting out a large surface of the earth for a time; but usually the figures succeeded each other too rapidly to be described even in shorthand, and at times they melted together in one long train of shapeless delicious emotion."

Now, can it be a "spirit of health," and not a "goblin damned," that comes in such a questionable shape as this? Disbelievers in revelation, who are unable to recognise its true nature by the light of psychology, will doubtless summarily reject the whole of the proffered news, and return to the messenger the answer which Festus gave to St. Paul. This we would affirm to be hasty, notwithstanding the suggestion conveyed by the information that "these inspiration-fits were of almost daily occurrence, and lasted from one hour to four or five hours," during which periods he "let the 'spirit' have its fling." He preserved a remarkably praiseworthy attitude, however, towards the phenomena; not believing that the "spirit" was any god, angel, or genius, but determined to devote all the time he could spare to study the matter with care, and for this purpose came to Europe and spent many months reading and meditating in seclusion in Paris.

The case just related is, indeed, a curious one, and we only regret that the facts furnished—especially of collateral evidence—should be as scanty as they are; were we to adopt the logic of chance, and count rather than weigh individually the members of the class to which the author's intellect belongs,

there might be little reason to rest dissatisfied with the first surmise that the story suggests, viz., that the periodic inspiration is only a peculiar form of epilepsy; and that having such a morbid foundation, it must be thoroughly impregnated with error, and must therefore give way, involving us in its destruction if we trust to it. We think, however, that this decision is not inevitable, but that after all it *is* an honest ghost that thus addresses us.

Next to actual disease, the cause next in probability would perhaps be some pseudo-disease, such as the influence of some drug, such as alcohol, opium, or haschisch; but the history of the case would persuade us to exclude any of these.

Is it, then, possible that the normal agency of the imagination can produce a state so closely bordering on delirium? A similar bounding state occurs, we know, in "the lover and the poet," within the range of health, as well as in "the lunatic;" but there are still other conditions, with which perhaps the Duke of Athens was unacquainted; perhaps the study of pure philosophy may be one, but certainly and chiefly the warmth of religious emotion cannot be passed over; for in this respect all prophets, oracles, and mystics attain, in their search for righteousness, an exaltation in nature similar to, but as much more lofty in aim than, the poet in his search for an ideal of humanity in nature, as the poet excels him who has only the common aims of action. And if, by a process of deduction, we are inclined to place our author within this class, his right to be there will be greatly strengthened by confirmatory *à posteriori* evidence; and this, we think, can be brought forward. The journey to arrive at our author's intellectual state is no less than an ascent of Olympus; and perhaps he will appear to have performed it on wings rather than on foot; but still can we not feel sure that he has only travelled along the ordinary path. It is hard to believe that such a mature intellectual product can have been formed quite unconsciously and delivered without any of the pangs of labour; but is not the account—meagre as it is that is given of his character habits, tastes and studies, a just description of what would be most likely to engender such an offspring? and that

the delivery should come suddenly and unexpectedly is not after all surprising, for it has been paralleled if not surpassed in abundance of recorded instances—notably in that of Jacob Böhme; and "unconscious cerebration" is unfamiliar to none. Still the question remains why the mind should make such upward reaches at all; and it is here, perhaps, that the analogous cases consequent on disease may afford some help; for such mental enlightenment is often a prominent feature in morbid psychology; it may be as well to note that this opening of the eyes does not imply an enlargement of the mind, being rather an analysis of the tissue of the mind—a separation of its web into its constituent fibres than a positive increase of mental substance. That it should occur in states of mental weakness (not loss), when the resistance is disproportionately large as compared with the power to overcome it, suggests that it may be a sort of compensatory process, perhaps an attempt to commence hypertrophy; and probably the plasticity induced by disease increases the facility for undergoing analysis. It would be interesting to know whether, in the case under consideration, there might not have been just a tinge of disease, showing itself, perhaps, by a slight degree of melancholia, sufficient to give some increase of plasticity. This seems to us to give some explanation of the reflectiveness of Hamlet, as well as of the illumination often occurring in epilepsy; but still there may be many other modes—perhaps an inverse one also—of the connection of genius with insanity.

In continuing his history, our philosopher tells us how, after much writhing in study, order at length began to show itself instead of the previous confusion; and his fully formed ideas burst their last adhesions. His train of thought was something as follows:—The subject-matter is mental, yet the vision is material; how can these, which are so opposed, be reconciled? He becomes convinced that the identity of mind and matter is real: it is acknowledged on all hands: do not all men—the vulgar as well as the inspired—speak of mind in terms of matter, as if it were endowed with size, motion, expressions, and other simple properties? On the other hand, there are undoubtedly differences—possibly only superficial—resolvable

essentially into these:—Matter is extended and dynamic, mind unextended and seemingly powerless: the difficulty of power disappears at once on reflection; for are not wishes, ideas, and feelings *motives?* The other fares no better, for extension is but externalisation, and this is only apparent, for at bottom what is extended consists of states of consciousness, as well as mind: perhaps, then, in time the mental world may expand into an analogue of the material world. Thus the grounds for separation disappear. Deferring, for want of light, the question of causal relation between them, he proceeded to ascertain *why* mind was described in terms of matter, and concluded that it was because the latter was the more familiar, just as less known material phenomena are described in terms of the more known: and if it is so difficult to describe material phenomena (e.g. geography, history) perfectly, how much more imperfect must our mental knowledge be without inspired vision! Common mental knowledge, then, could be much improved by adoption of an improved system of symbols, and this (which he obtained by inspiration) he purposes to give. For, though he admits that many a mystic before has had sufficient mental knowledge, the expression of it has been prevented for want of more complete material knowledge—mystics as a rule being ignorant of, and indifferent to, common science. Admitting that even inspired vision gives but fragmentary perception of the assimilation of mind and matter, he holds that as they resemble each other in some degree, there is reason to believe they do so in all. His keenest perception of this identity, the homology being complete, was that of observing a peculiar formative principle—best known as life—actuating both; thus each is an organism. Nearer the truth than this animistic philosophy it seems to us impossible to go. Thus mind as well as matter is governed by inherent impersonal laws: the difference is one of degree: one is much consciousness to little substance, in the other the proportion is reversed. Mind is what matter has been, matter what mind will be. (Here he states the doctrine of evolution, but from an idealistic point of view). Having now attained the climax of his knowledge, he paid his attentions to the lower regions of physical science, to verify his material conceptions, and apply these material laws

and their vocabulary to mental phenomena, as if by geometrical superposition. Nothing then remained but to give expression to his theory: he apologises for its rough style, which certainly exists, and there is not freedom from ambiguity, but this perhaps does not extend much below the letter. He claims excuse on the ground that the " moral law has never before been made a subject of exact demonstration from common experience," which we think can scarcely be allowed while Spinoza's Ethics is in the memory.

But we must give a short outline of his exposition; it is divided into chapters more or less dependent. The first is upon the Fallacy of Language—in which he assails, first by induction then by deduction, the current "rhetorical theory," that language, being such an imperfect symbol, can convey actual knowledge: at first, disproportionate space appears allotted to this subject, but the object seems to be to emphasise the fact that a thing and its symbol are not identical.

Next is set forth the Essentials of Logic; but this plan seems to fall short of what we should expect of a "system of logic from the vitality standpoint," which would suggest something more Hegelian than the Associationist theory on which it is chiefly based; it is rendered as precise as possible by the use of geometrical diagrams. " Substance " is declared to be the essential attributes of a system. Abstraction and conception are then treated in more detail: causation is resolved into invariable succession. Some specification of the theory is carried out in a list of definitions, and various corollaries in the form of notes.

The author's opinion has now become decided as to the causal connection of mind and matter, stating definitely that the former is the invariable antecedent. This is proved by the incontrovertible intuition of the mental source of human expression; now as human acts are identical with not-human acts, the latter also must own a mental source. He is careful to avoid the error of anthropomorphism by showing that that crude doctrine arises from imperfect abstraction of " soul " from " human soul." He seeks confirmation in the etymological opinion that scientific terms have been derived by analogy from human phenomena.

He has now commenced a rigid logical chain. The next link is Interpretation—a special form of inference from the foregoing conclusion, showing how we learn the causes of things by applying human causation. He next exposes the fallacy of Materialism. It is another deduction from the same: he lays down as an axiom that man can have no perfect knowledge of anything but man, and the more objects resemble man, the better can they be known: much like the dictum of Protagoras that "man is the measure of all things." Therefore the universe cannot be deduced from purely material induction—which would be to explain the more known by the less known. And though he allows that it is impossible to conceive the mental causes of not-human individualities, yet it is still possible to formulate a sufficient theory of the universe.

The chapter on "Symbolism" is interesting ground; as it deals with principles that have such wide and familiar application, being the law of formation of "Aberglaube," whether springing up out of religious emotion or poetic frenzy, or as the delusions of insanity; besides prompting the numerous ceremonies of social life (*vide* Spencer's 'Ceremonial Government'). And thus we see that "material science is not the final wisdom, though absolutely necessary for two purposes—the perfection of our material civilisation, and the expression of our mental knowledge—the latter being accomplished by our arts and industries, which are more or less conventional." A long and various catalogue is given by way of illustration. "The perpetuation of symbols after they have ceased to be effective, is superstition." But symbols have the disadvantage of being proved of less homogeneous abstractions, and less true assimilations than the facts they represent. As a conclusion, he lays down that our mental knowledge cannot be expressed except by material symbols, but that as a "supreme mystery," the best of these consist of *the highest generalisations of physics. chemistry, and biology* Of course, he states expressly his theory of the universe, starting with the postulates of three ultimate principles (corresponding in function with Aristotle's Causes), viz., Substance, Motion, Order; by whose combination the whole mass of phenomena is produced. Substance appears in at least three grades—

Matter, Soul, Spirit—arranged concentrically, as it were, the last being inmost; but he says there may be an infinity of other modes within the last, as he cannot fix on an absolute centre: this possible numerical infinity gives the doctrine as a system less apparent firmness than, though it has much of the symmetry of, Hegel's, which claims a foundation on ultimate fact. Motion consists essentially in attraction. Order is best witnessed in the form of evolution; but it is hard to see why it should be distinguished altogether from motion.

As the crown of the universe comes the human system, which is treated of in the final and most characteristic chapter. It is, of course, shown to be only a special case of the preceding: it consists of a bodily organism to produce vital phenomena, a mental organism (soul) to produce the mental, and to observe the vital phenomena; and then a spiritual organism (spirit) to produce spiritual and observe the mental phenomena. Perhaps there is a partially formed organism within to observe the spirit, and possibly an infinity of germs arranged concentrically within this, like the infinite number of germs within an ovule, but which never as yet have attained actuality.

Then an analogy is traced between the vital and the mental organism, of especial interest to students of insanity being a paragraph (§ 14, p. 106) containing a description of the analogies of morbid states, on the principle that "the soul, like the body, is happy or miserable according to its state of health;" the modes of its application appear mainly correct. The ethical application is not neglected; for "the individual soul belongs to a great mental system." He deduces from his principles the strange ethical rule, "Classify yourselves rightly," as the best guide for practice. Some exception however must, we think, be taken to it, as long as it neglects an ideal of conduct. Perfection, according to his theory, is religion—the growth and life of the spirit; and has been variously spoken of as Nirvâna, the Great Awakening, being born again, absorption into the Deity, entrance into the Kingdom of Heaven, &c. Satan is a personification of Destruction —especially Spiritual Destruction.

After this sketch of the system, curiosity is awakened to

assign him a definite position among the constellations which the various philosophical schools form. And, as is the case with the authors of so many other systems, he seems to belong so much to all and yet so little to any of the rest, that it is somewhat difficult to fix his exact latitude and longitude. He seems also curiously disinclined to own allegiance to any of his great predecessors, however much the framework of his system may resemble theirs; though also he states more or less explicitly that he has read extensively—especially of the mystical writers, and indeed occasionally exhibits it by quotation—yet he seldom draws attention to the resemblance, either for more clearly expressing or for confirming his own views. Of all the chief systems, his reminds us most of Berkeley's; for just as the latter, taking vision as his theme, proceeds to construct an all-comprehensive theory, of which phenomena are the signs by which it is known to us, so does our author form a somewhat similar theory, starting from language as the proximate evidence of a subjacent reality: moreover, the goal of each is not widely remote; for though D. M. is no professed apologist for a Deity, he is scarcely a step from Pantheism.

And thus, though perhaps no single element of the work can be regarded as absolutely original, and the whole is rather a new turn of the philosophical kaleidoscope than a perfectly new piece of architecture, and is even rather bare in some important factors, such as the immensity and penetration of a Kant, and the richness and eloquence of an Emerson, yet the clearness of simplicity, the impartiality, and, above all, its connection with the circumstances of its genesis, make it one warmly to be recommended.

F. L. Benham, M.B.

Heidenhain on Animal Magnetism. ('Animal Magnetism, Physiological Observations,' by Rudolph Heidenhain. Translated from the 4th German edition, by J. C. Wooldridge. London: Kegan Paul & Co. 1880.)

"Animal Magnetism" seems to be entering on a new phase, that of strict scientific investigation. This is a matter of con-

gratulation, for the extraordinary character of the phenomena described under this term suggests questions, a solution of which must have an immense influence on physio-psychology. Though—as Mr. Romanes, who writes a preface to the translation, justly observes—Braid of Manchester, forty years ago published the results of investigations on this subject, which anticipated almost all that has been recently apparently rediscovered by Heidenhain, yet they did not succeed in exciting the attention they deserve from men of science, and "animal magnetism" has been left to quacks and mountebanks. Mr. Braid's works are likely now to be studied with a different interest, and we are glad to learn that they are about to be published again in a collected form.

Among other recent researches relating to hypnotism and allied states may be mentioned those of Czermak and Preyer, on the state into which some of the lower animals—especially fowls—can be thrown by fixing this attention in various ways; a condition for which Preyer has coined the name "cataplexy." The researches of Charcot at the Salpétrière on hysteria have also excited much interest and speculation.

It is the advantage of Heidenhain's work that the observations have been made on intelligent individuals in normal health, and, as far as that can be determined, reliable integrity; while the investigator himself has a name as a physiologist which at once vouches for the *bona fides* of the facts and methods employed for establishing them.

The memoir consists of two parts: the first, a semi-popular lecture, delivered at the beginning of this year; the second, a further series of observations, made in conjunction with Dr. Grützner, and of greater scientific precision.

It need scarcely be said that "animal magnetism," though it gives the title to the book, is used only as a term better known than "hypnotism," which was introduced by Mr. Braid, and adopted by Heidenhain throughout.

For the production of hypnotism there is no question of specific force on the part of the experimenter. Hypnotic experiments, however, do not succeed in every one's hands, or on all on whom they are tried. The susceptibility depends on the existence of, as the author terms it, a greater or less

degree of "sensory irritability," and, according to him, pale, anæmic individuals are most liable to hypnosis. Hansen, on the other hand—whose public exhibitions in Breslau originated Heidenhain's investigations—thinks active, muscular individuals are more susceptible; and therefore prefers the active Englishman to the phlegmatic German.

Whether the necessary "sensory irritability" exists primarily or not, the method first followed is to make the individual stare fixedly for some six or eight minutes at a shining faceted glass button, or other glittering object, the eyes being made to converge upwards as much as possible. This was introduced by Braid, but apparently the Egyptian conjurors produced the same effect by making the hypnotee stare at cabalistic signs on a white plate. In some persons the staring at a glass button for several minutes is all that is required to induce hypnotism, but usually manipulations or passes are required in addition.

The effect of staring fixedly for some time is, according to Heidenhain, to produce a condition of irritability which favours the establishment of the hypnotic state, just as excitement of the public mind generally increases the number of susceptibles. When the staring is kept up there is a feeling of dazzling lachrymation, dimness, and contraction of the field of vision, and the button seems to swim. Then follow spectra, and after images.

The manipulations or passes which are in most cases required in addition are merely weak, continued, and monotonous stimuli of the skin, or of the sense of hearing. The individual who makes the passes, or the "magnetiser," may even be dispensed with, provided the requisite monotonous stimulation is produced. Hence the mere ticking of a watch will hypnotise some. The cutaneous stimulation induced by the "passes," which consist in slowly moving the hands close to the hypnotee's face from forehead to chin, is a compound effect, partly tactile, by the draught of air set up, and partly sensations of temperature. A curious feeling is thus induced in the skin, though it is not actually touched. Some hands are more effective than others in thus stimulating the skin, and hence some people may fail because they have not the

warm moist hands which are found best, and they are not altogether uniform and steady in their movement. For the hypnotic condition is dispelled by strong, sudden, or irregular stimulation of those senses, stimulation of which in the reverse manner lulls consciousness to sleep. Thus cold, a touch, blowing on the face, a loud sound, a bright light falling on the eye, &c., are all sufficient to wake the hypnotee. A change in the direction of the passes, or a mere change in the retinal picture, by one operator taking the place of another, will also cause awakening.

In very susceptible individuals the hypnotic condition can be induced very easily, especially by acoustic stimuli, and even against the will of the individual. In some the idea of approaching sleep is enough to bring it on. Thus it is possible to make use of a specified hour, or some object previously agreed on at which the individual is to look, to induce hypnotism at any imaginable distance between the operator and medium. All that is required is the induction of the idea that sleep is coming on.

The first demonstrable symptom of the hypnotic condition is a spasm of the mechanism of accommodation, so that the "far" point approaches the "near" point. After some time also in most cases the pupils enlarge, the eyes open more widely, and the eyeballs protrude. There are almost infinite variations in the degree and manifestation of the phenomena of hypnotism, psychical and physical. The most numerous cases are those in which the incapability of opening the closed eyelids is the sole abnormal phenomenon. Next to these come a large number of people who cannot, or can only with great difficulty, open their mouth when it has been closed. There is spastic contracture of the orbicular muscles. In other persons the spastic condition is more or less general all over the body. Consciousness may, however, be completely retained in these cases, or only slightly reduced, demonstrable by some defects in the recollection of what has occurred.

In the types just mentioned the muscular phenomena form the chief, or at least the most prominent, features; in another series psychical phenomena prevail.

As regards the muscular system there appears to be an

increased reflex irritability, and tendency to tonic spasm. More or less marked cataleptic rigidity ensues, so that the limbs will remain fixed in whatever position they may be placed. Volition can be exerted only with great difficulty, and not unfrequently, instead of simple contractions, convulsive movements are set up which may become more or less generalised. By reflex irritation, such as gentle stroking of the skin, the underlying muscles can be thrown into tonic contraction, and with a greater increase of excitability irradiation occurs, following certain definite lines with remarkable uniformity.

Stroking the ball of the thumb causes adduction of the thumb. If this is increased, the muscles of the forearm, especially the flexors of the fingers, are thrown into contraction. The other movements of the arm are still, however, free. With further increase of the irritation, the whole of the muscles are thrown into tonic spasm, and the limb becomes stiff throughout. But in still higher degrees of irritability the spasm becomes still more extensive, and, as demonstrated by Heidenhain on his brother, the following was the march of the spasm: Left thumb; left hand; left forearm; left upper arm and shoulder; right shoulder and arm; right forearm; right hand; left leg; left thigh; right thigh; right leg; muscles of mastication; muscles of the neck.

Similarly, if the right foot is irritated, the spasm ascends the right leg, then descends the left leg; then ascends the right arm, descends the left arm; and ultimately invades the muscles of mastication and the cervical muscles.

A further investigation of the areas of reflex excitation led to the following curious localisation:

Stroking of the skin at the side of the spinous processes of the upper dorsal vertebræ caused elevation of the arms, with simultaneous slight flexion, so that the hands tend to meet above the head.

Stimulation of the lower dorsal region caused contraction of the latissimi and rhomboidei, with strong backward movement of both arms and simultaneous flexion.

Stimulation of last dorsal and first lumbar vertebral region caused spasm of the whole erector spinæ, with elevation of ribs (*lev. cost.; intercost.*), without contraction of diaphragm.

Stimulation over the lower lumbar vertebræ and sacral region, the person being seated, caused contraction of the flexors of the leg; then of the ileo-psoas, and hence (the thigh being fixed) bending of the body forward on thighs.

Stimulation of the sternal region caused contraction of the pectoralis major, with adduction of arm, and simultaneous action of the extensors.

If while the hypnotised individual is standing, the skin is stimulated at the side of the last lumbar vertebra, so as to produce a dragging downwards of the skin, the leg of that side moves backwards. Alternate stimulation of the same area on the two sides causes the individual to walk backwards with short steps.

If one side is stimulated successively, the leg moves backwards each time, till the legs are separated to their extreme limit as in spanning a broad ditch.

In muscular individuals the rigidity which can be induced throughout is so great that the whole body becomes as stiff as a board, and will remain supported merely by resting on the two ends.

In individuals who have been hypnotised the increased reflex irritability of the muscles lasts for some time, but it ultimately disappears if the experiments are not repeated for some time.

Next as to the psychical phenomena. In many persons, more or less profound sleep, mostly but not always accompanied by analgesia, alone occurs. Muscular phenomena may be totally absent during the condition of sleep, or there may be more or less developed catalepsy. In others, and those form the most interesting class, the cerebrum does not sleep as a whole, and the peculiarity occurs that during this state impressions made on the organs of sense are still able to excite correlated and co-ordinated muscular movements. Of these the numerous forms of "imitation automatism" are remarkable examples.

Movements carried out before a hypnotised individual, who apparently has his eyes shut, are nevertheless perceived by the eye, as the closure of the lids is not complete. "The perceived, but not consciously perceived, movement is imi-

tated. The same, with many movements, which are accompanied by a familiar and distinctly audible sound. I clench my fist before Mr. H——, who stands hypnotised before me; he clenches his. I open my mouth; he does the same. Now I close my fist behind his back, or over his bent head; he makes no movement. I shut my mouth, still over his bent head, rapidly, so that the teeth knock together; he repeats the manœuvre. I noiselessly contort my visage; he remains quiet" (p. 11). The optical or auditory impression liberates movements which have the type of voluntary movements, but are really not so. Sneezing and coughing are not imitated by hypnotised individuals; nor are laughing or singing. At most only facial contortions, or some of the associated movements, are carried out. The extraordinary imitative tendency of the hypnotised individual explains his apparent subjection to the will of the experimenter. In a loud voice the medium is ordered to do a certain action. Even though he may have no idea of the nature of the command, yet the performance of the action by the experimenter compels its imitation by the medium.

People who are at first somewhat intractable as regards imitation improve by continuous repetition of the movement before them, and the imitation often becomes marvellously exact even to the smallest detail. Passive movements communicated to a medium are as a rule continued by him, so that if he is made to walk he goes on readily. As a rule the medium does not remember, or at least cannot recall, what has occurred during his hypnotic condition; but that the impressions have been made is shown by the fact that with more or less suggestion what has been uttered may be brought up again in his mind.

A further very extraordinary series of phenomena, observed first by Berger, is described under the head of "speech automatism." Berger found that on applying pressure to the neck of a hypnotised individual, over the spinous processes of the lower cervical vertebræ, he could often be induced to repeat words spoken in his hearing; it being a matter of indifference whether the words were intelligible or not. The repetition is made in a very monotonous tone, which varies in individuals.

Some talk in a hollow sepulchral tone, others more softly, almost lisping; but the same person always repeats in the same way.

According to Berger, the hand must be warm; but Heidenhain finds the pressure of a cold hand just as effectual.

The following up of Berger's speech experiment led to the discovery that most persons who repeat when pressure is made on the neck, repeat also when one speaks, either simply, or better through a speaking trumpet, towards the neck without any pressure whatever, while they remain dumb when one talks towards any other part of the head, or even into the ear.

They repeat very readily and distinctly when the experimenter's speech is directed to the epigastrium, less distinctly when it is directed to the larynx, or through the open mouth to the back of the pharynx. If a vibrating tuning-fork is applied against one of these sensitive parts, a note like that of the tuning-fork is pronounced, while if the fork is held near the ear the medium often awakes with an expression of pain, and states that he has had a feeling of burning in the ear.

If by means of the tuning-fork the limits of the epigastric area are mapped out, it is found that it extends from about two finger-breadths below the sternum for about two inches downwards, and for about the same distance on each side of the middle line, while the lateral abdominal, umbilical, sternal and costal regions are all absolutely insensitive.

The sensitive epigastric area corresponds to the anterior wall of the stomach. And since the stomach, larynx, and pharynx all derive nervous supply from the vagus, the speculation is made that in some way the vagus conveys sound vibrations to the vocalising centre. It is further supposed that there may be anastomoses between the vagus and lower cervical nerves; but in a footnote (p. 64) it is stated that other sensory nerves besides the vagus may convey the sound vibrations, though the vagus seems more especially so adapted. These results are certainly of a very extraordinary character.

Under certain conditions hypnotised individuals also obey the command to carry out certain actions, and it is possible to

induce dreams by talking into their ears. In the dreaming state they readily reply to questions put to them, and carry out movements in accordance with their dreams. At first Heidenhain did not succeed in inducing these phenomena, but ultimately he was able to produce this "automatism at command" without difficulty. To obtain this it is necessary that the hypnotism should be less deep than for the imitation automatism, and this it was found could be brought about by laying the hand on the medium's head, a proceeding which has more or less tendency to cause awakening.

The character of the acts done at command is a guarantee of their genuineness, as they are such as would never be done by the individual in his senses. He acts in accordance with whatever suggestions are made, and exhibits the emotions proper to the conditions under which he is supposed to be placed—occasionally with unpleasant intensity. On awaking the individual either has no recollection of what has occurred, or can recall it more or less imperfectly with the aid of hints and suggestions.

The command automatism is a much more complex process than the imitation or speech automatism, for in these certain actions made by the experimenter induce similar actions in the medium. In command automatism, however, dissimilar actions are called forth—words calling forth actions. In those cases where the actions at command are not in immediate relation with some acted dream, commands are only effective in reference to very simple every-day actions rendered almost mechanical by repetition. The command must be *direct.* Thus such a sentence, "I should like to know the time" has no effect on the medium; but when he is told, "Show me your watch," he at once obeys. The command must also be *complete.* "To the door," causes no action; but "Go to the door," causes the medium to walk, not necessarily to the door, but away from his position, as if the command had been merely "Go." When requested to pass something, the medium lays his hand on anything that comes in his way, unless he is asked for something he is quite familiar with—say, in his own pocket.

Another series of phenomena, full of suggestive interest, is described under the head of "Unilateral Hypnosis."

In some persons stroking of one side of the head produces a paralytic condition of the opposite, or, it may be, of the same side of the body. The individual to be tested is made to sit down and the parietal region gently stroked or pressed with the warm hand. In those predisposed this induces first a weight in the arm, and then more and more complete paralysis. When he makes a violent effort to raise the limb spasms occur in it. In the case of Herr Heidenhain stroking of the left side of the head caused paralysis of the right arm and leg, and paralysis of the right side of the face—stated to be like paralysis of the facial nerve (?).

The medium at the same time is said to be aphasic, at least he cannot pronounce or repeat words. When the right side of the head is stroked in a similar manner all the phenomena appear on the left side, with the exception of the aphasia. If both sides are simultaneously stroked all four limbs become cataleptic, but no disturbance whatever occurs as regards speech or the facial movements.

If the left side be first stroked, producing right-sided paralysis and aphasia, and then the right side also, the aphasia disappears, aud the limbs of both sides become affected. If, however, the left side is first stroked, and then the right, stroking of the left being omitted, the aphasia and right-sided paralysis disappear and left-sided catalepsy takes its place.

Measurement of the volume of the cataleptic arm by Mosso's volumeter shows that the blood volume is greatly reduced, as compared with that of the other. The phenomena are reversed when the cataleptic condition ceases.

In one case, Mr. W——, unilateral stroking caused catalepsy on the same side. The aphasia can only be induced in him by stroking the right side of the head. Berger, who has also worked at unilateral hypnosis, thinks that stroking the forehead causes crossed catalepsy; stroking the temporal region, catalepsy on the same side. The authors attribute the disturbance of speech to "contracture," or spasm of some of the muscles of articulation—especially the laryngeal—though the mouth can be opened and the tongue moved freely.

It is to be especially noted, in reference to unilateral hypnosis, that consciousness is not perceptibly affected.

But there are very remarkable disturbances of sensibility, more particularly investigated as regards vision, which are worthy of special attention. These have been carefully examined by Cohn, in conjunction with Heidenhain and Grützner.

It may be remarked also that Heidenhain has noted defective perception of temperature on the affected side, which, however, he has not further followed up.

As regards vision, the disturbances are spasm of accommodation and colour blindness, confined to the cataleptic side.

The accommodation spasm is so great that the "near point" is only twenty millimètres from the eye.

The condition of the eye as to colour perception corresponds to that given in Stilling's new Atlas (Table IV., b. l. 1, 2).

All colours appear different shades of grey—from dirty dark grey to a clear silver grey.

Though the eye is thus insensitive to colours, pressure on the eye or variation in pressure causes subjective colour sensations.

If the hypnotised eye has been kept covered for some time and then a coloured disc looked at, the disc appears coloured, not, however, with its own, but its complementary colour, e.g., a red disc appears green. But directly after this it appears grey, just as it does if the eye has not been kept closed.

Under the influence of atropin the phenomena change. Red and green still appear as different shades of grey. Blue and yellow, however, do not appear grey, but differently at different stages of the atropin action. First, yellow appears grey with a glimmer of blue. Second stage, yellow appears pure blue. Third stage, yellow appears blue with a slight tinge of yellow. Yellow is seen, as it were, struggling through a blue mist. Fourth stage, yellow appears mostly yellow, with a slight tinge of blue. When blue is tried the corresponding result is obtained, the blue with a slight yellow tinge is seen. During the action of atropin, yellow or blue passes from grey through the complementary colour to the true colour, while red and green appear only as different shades of grey.

It is suggested that as atropin removes the spasm of accommodation and also the colour blindness, there is a causal relation between the two phenomena. But on the other hand, as

physostigma produces spasm of accommodation, leaving the colour sense quite unimpaired, and as in many hypnotics accommodation spasm occurs without colour blindness, it is argued that atropin has a direct action on the elements concerned in colour perception.

The colour blindness produced by stroking one temporal region disappears by stroking the other side, just as in the aphasia similarly induced. Another curious fact ascertained by Cohn is that a person naturally colour blind, when unilaterally hypnotised, was able to distinguish colours which were otherwise undistinguishable.

It has been stated that in unilateral hypnosis the sensorium is apparently unaffected—indicating, it is argued, that one hemisphere of the brain fully suffices for all psychical functions. But more exact observation shows that there is a certain disturbance in the connection between sensations and movements on the hypnotised side.

In a person hypnotised on the left side, there is a certain diminution in the fluency of writing with the right hand, though the hand is otherwise quite movable. The handwriting acquires quite a foreign character. The letters are very close together, and not unfrequently a letter is reversed. It also appears that there is a very great tendency to imitative movements with the right hand during the unilateral hypnotic condition of the left, a tendency which in the normal condition does not exist. The involuntary imitation seems to be excited by the eye of the cataleptic side, for the imitation ceases when this is closed. From this the conclusion is drawn that the movements of each arm are influenced by both hemispheres.

Such are the main facts of this interesting and concise memoir, and they are such—admitting them all to be genuine, and this character they seem to have beyond all similar statements—as to justify the belief Heidenhain expresses, "that we have in this method a means of investigating the functions of the brain, the place of which can be supplied by no other method of investigation."

The hypotheses he offers to account for the phenomena, however ingenious and suggestive, have to us at present less value than the question of the bare facts themselves. Specula-

tion is easy, and we have had it *ad nauseam;* but speculation to be of any value must be strictly subordinate to a more thorough investigation of the pathological conditions. This inquiry comes specially within the province of physicians, who will see in many of the facts above mentioned suggestive resemblances with the phenomena of cerebral disease, functional and organic.

The hypothesis advanced by Heidenhain is that the phenomena of hypnotism are due to inhibition of the activity of the ganglion cells of the cerebral cortex; the inhibition being brought about by gentle prolonged stimulation of the sensory nerves of the face, or of the auditory or optic nerve.

That the lower centres, those of equilibration, &c., are active, seems the rule, though occasionally, during hypnotism, these give way also, and the individual falls down. The affection of the cortex explains why hypnotised individuals neither acquire conscious perceptions as the result of sensory impressions, nor spontaneously make voluntary movements.

"Normally when the idea of a movement presents itself to our consciousness, we can carry that movement into effect or not. In the hypnotic condition, owing to the absence of the inhibitory power of the will, the unconscious perception of the movement irresistibly brings it about—a process in all respects analogous to reflex action."

The inhibition of the cortex is not due to anæmia reflexly produced by contraction of the cerebral blood-vessels. An examination of the retina by Förster did not reveal any indication of this; and more convincing perhaps is the fact that hypnosis could be induced in one person during the action of nitrite of amyl, which enormously dilates the blood-vessels of the head. Arguments are adduced to show that the inhibition depends on irritation of certain sensory nerves, similar to the mechanism of reflex paralysis.

DAVID FERRIER.

Clinical Cases.

A CASE OF PROGRESSIVE MUSCULAR ATROPHY WITH UNILATERAL ATROPHY OF THE TONGUE.

BY BYROM BRAMWELL, M.D., F.R.C.P. EDIN.

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In the course of progressive muscular atrophy, and especially towards its later stages, it not unfrequently happens, as we all know, that the degenerative process which primarily had invaded the large nerve cells of the anterior cornua of the spinal cord, extends upwards and involves the motor nerve nuclei of the pons and medulla.

In such cases the characteristic symptoms of glosso-labial paralysis are added to those of the original affection.

In the following case of progressive muscular atrophy the degenerative process had extended to the medulla and pons, and had seriously affected the nerve nucleus of the 9th (hypoglossal), and to a less extent that of the 7th (facial), on the right side, the result being most marked atrophy of the right half of the tongue, and some wasting of the right cheek. There was no affection of speech, no difficulty in swallowing, in short, no symptoms of glosso-labial paralysis. All the movements of the tongue could be accurately performed; indeed, until I directed the patient's attention to the condition of the organ he was not aware that there was anything the matter with it. This fact, and the circumstance that the lesion in the medulla was so limited and so well defined, must be my excuse for reporting the case in what may perhaps be thought somewhat wearisome detail.

M. B., æt. 55, labourer, living in the Cowgate, Edinburgh, was sent to me by my friend Dr. Wilson, at the beginning of March of the present year, and was subsequently, at my request, admitted to the Royal Infirmary, Ward 23, where through the kindness of Professor Sanders I had repeated opportunities of examining him. I have also to thank Professor Sanders for allowing me to make use of his

notes, and Mr. Frank Rand, the clinical clerk, for valuable help in the examination of the case.

The patient's chief complaints were muscular weakness, most marked in the upper extremities, especially in the right; numbness and increased sensitiveness to cold in the right half of the body; and giddiness.

Previous History.—Ten years ago he began to feel his right hand and arm "numb and weak." The weakness slowly but gradually increased. After a time (the exact date he was unable to fix) the left arm and hand became similarly affected. Until two years ago he was able to follow his ordinary occupation, that of a labourer. The weakness then (two years ago) became so great that he had to take to chopping sticks. For the past year he has not been able to follow any employment. Ever since the muscle weakness was first noticed he has been subject to attacks of "lightness in the head." These attacks have been much worse of late, and he has in consequence had several falls.

He knows no cause for his complaint. His general health until lately has been particularly good. He has been fairly temperate. Has not had syphilis. Like most labourers, he has been a good deal exposed to wet and cold, but has never suffered from rheumatism. His surroundings until lately have been fairly comfortable.

Family History.—Good. His father and mother died at an advanced age. His brothers and sisters (five in number), and his own children, are all healthy. So far as he knows, he has no hereditary tendency to nerve complaints.

Present Condition.—He is a small man, and looks much older than his years. Is very feeble, and walks with difficulty by the help of a stick, the gait being tottering and senile. He has a marked stoop.

There is a recent scar over the right eyebrow, the result, he says, of a fall during an attack of giddiness.

Nervous System; Motor Functions.—The muscular power generally is very much below par. The weakness is extreme in the upper extremities, especially in the right. The patient is unable to "spread" the fingers of the right hand. The movements of the phalangeal joints of the fingers and thumb of the right hand are imperfect. This is in great part owing to joint stiffness. All the other movements of the right upper extremity can be performed, though with extreme feebleness, the slightest passive resistance being sufficient to prevent their execution. The grasping power of both hands, as measured by the dynamometer, is much below par, that of the right being much weaker than that of the left.

The right hand presents to some extent the "bird-claw"

character, but the appearance is not typical, owing to the fact that the fingers and thumb are rigid at their first phalangeal joints.

The rigidity of the little finger dates from childhood, and is the result of a fracture; that of the ring-finger was caused by an abscess some few years ago; that of the middle finger resulted from an injury thirteen years ago; that of the thumb dates back twenty years, and resulted from an injury; that of the forefinger came on of itself, without any obvious cause. (? Could these injuries have been in any way the cause of the disease? Could they have been the exciting cause of an ascending degeneration, which primarily affected the nerve nuclei supplying the hand muscles?)

The fingers of the left hand are slightly flexed at their first phalangeal joints. There is some stiffness and rigidity on passive movement at the same joints.

All the small muscles of the right hand and thumb are in an extreme state of emaciation. The muscles of the right forearm and the muscles attaching the right upper extremity to the trunk are also much wasted. The atrophy is more marked in some muscles and in some parts of muscles than in others; the upper part of the trapezius, for example, is much more affected than the lower.

The small muscles of the left hand and thumb, and of the left upper extremity, are also atrophied, though in a less degree than those of the right.

The right half of the tongue is much wasted; it is soft and flabby, marked with furrows and wrinkles, and presents a striking contrast to its plump fellow of the opposite side. The atrophy is accurately limited to the middle line. The tip of the organ when fully protruded is slightly turned to the right (atrophied) side. All the movements of the tongue can be accurately performed; in fact, as I have already remarked, until the patient's attention was directed to the condition of the organ he was not aware that there was anything the matter with it. The soft palate and uvula are natural. There is no difficulty in speech, mastication, or deglutition.

The right side of the face is somewhat atrophied, the wasting being most marked in the cheek, which feels thinner and less firm than the left.

The other muscles supplied by nerves coming off from the medulla and pons seem normal. The muscles of the trunk, with the exception of those before mentioned, and the muscles of the lower extremities, are soft and flabby, but do not present any appearance of special atrophy.

The *mechanical irritability* of the atrophied muscles seems natural. *Electrical irritability,* a careful examination of the

atrophied muscles, including the tongue, show a simple diminution, especially in the strength of the contraction produced, to both forms of current. There is no trace of "reaction of degeneration;" even in the muscles which seem most atrophied a contraction can be obtained by a strong faradic current. Very marked fibrillary twitchings frequently occur in the atrophied muscles, including the right half of the tongue.

The *reflexes* (superficial, deep, and organic) all normal.

Co-ordination seems normal.

The *muscular sense*, as tested by weights, appears to be normal.

Sensory Functions.—He complains of feeling cold. Says that the left side of his head and the right side of his body are numb.

Tactile sensibility and the *power of localising* impressions are natural.

Sensibility to pain is natural.

Sensibility to temperature is imperfect. He can always distinguish cold objects, indeed sensibility to cold is increased, but he not unfrequently says warm objects are cold. At other times a pretty prolonged contact of a warm object is necessary before it is recognised as such (delayed impression).

Sight.—The left eye is blind, the result of an old injury. Sight in the right eye is fairly good. Nothing of importance was detected on ophthalmoscopic examination.

Taste is natural.

Smell is natural.

Hearing.—Aërial sounds are very imperfectly heard in the left ear; the skull sounds, as tested by the tuning-fork, are well heard on both sides.

Intelligence is good. The patient sleeps well, and with the exception of occasional attacks of giddiness feels nothing wrong with his head. The attacks of giddiness are of short duration, and generally occur when he gets up or turns round quickly. He does not vomit, and has never had a fit.

The *temperature* is normal or *subnormal.*

The *radials* are somewhat hard and rigid; the heart is weak; and there is slight bronchitis. But with these exceptions the other organs and systems are normal.

The patient remained for some time under observation in hospital. His general health improved considerably. The muscular condition underwent no obvious change. He used to like faradisation, thinking it did him good. When last seen (Oct. 7th) he was much *in statu quo.*

COMPOUND FRACTURE OF SKULL—COMPRESSION OF BRAIN BY CLOT BETWEEN THE SKULL AND DURA MATER—RECOVERY.

UNDER THE CARE OF T. PRIDGIN TEALE, M.A. OXON., F.R.C.S.

T. S., aged 28, a groom, was kicked on the head by a recently shod tram-horse, on the morning of October 17th, 1879, and was at once admitted into the Leeds Infirmary.

Condition on admission, at 10.30 A.M. There was a wound about two inches in length, situated three inches behind the right frontal eminence, and just in front of a line drawn from ear to ear. The skull was fractured, and a large piece of bone was depressed, and there was free hæmorrhage from within the skull. Pulse 55. Respiration 12. Patient comatose.

Having enlarged the wound, Mr. Brown, the house surgeon, removed the depressed portion of bone. Immediately after the bone had been raised, the patient opened his eyes and felt greatly relieved.

At 11.30 the pulse was 88, and there was free bleeding from the wound.

Oct. 18th. Day after the accident. There is a loss of motor power in the left arm, gradually increasing, but without loss of sensation. Left leg natural. Patient conscious.

Oct. 20th. Patient's symptoms worse. Inability to answer questions. Left hemiplegia complete. Temperature at 4 P.M. 104°·3. Pulse 60. Incontinence of urine. A large clot in the wound. He was seen by Mr. Pridgin Teale at 11 P.M. He was then unconscious. Mr. Teale, believing that his symptoms were due to compression of the brain by a large, firm clot occupying the site of the removed bone and extending beneath the skull for some way, but external to the dura mater, broke up with the finger the clot, but did not remove it as it was firmly adherent to surrounding structures. In six hours the patient's temperature became normal, and he was more conscious.

Oct. 21st. More indications of returning consciousness.

Oct. 22nd. Answers questions. Has regained power over the bladder. Is still hemiplegic.

Oct. 25th. Hemiplegia disappearing.

Nov. 10th. Hemiplegia quite gone.

Henceforth he made good progress, and was discharged apparently sound, Feb. 8th, 1879, and is now, Sept. 1880, well and following his employment as a groom.

This case is interesting. First, as showing the direct results of compression by depressed bone, immediately relieved by removal of the compressing causes. Secondly, in the gradual development in detail of symptoms of unilateral pressure by blood external to the dura mater, and not in an absolutely confined space, the pressure being due to the "setting" of the clot into a stiff mass. Thirdly, in the immediate commencement of retrocession of the symptoms, as soon as the cohesion of the clot had been broken up. (Mr. Teale did not think it well to scrape away with instruments the firmly adherent clot, lest the hæmorrhage should be reproduced.)

The order in which the symptoms improved is well shown. 1st. The fall in temperature. 2nd. Return of consciousness so as to understand what was said to him. 3rd. Ability to answer questions, and recovery of power of micturition. 4th. Slow recovery of power of leg and foot. The first symptom of the onset of the second period of compression was loss of motor power in the left arm.

ON FUNCTIONAL ATHETOSIS AND INCOORDINATION OF MOVEMENT.

BY LEWIS SHAPTER, M.D. CANTAB.

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J. P., aged 7 years, thin and delicate, but intelligent in appearance, was admitted into the Devon and Exeter Hospital on the 20th of February, 1879. There appeared to be a general tendency to tonic spasm of the flexor muscles of the left side of the body, as evinced by distortion of the hand and foot, on movement and at rest; an inability to keep the left side of the body in any one position for any length of time; and a strained incoordinate movement of the arm and leg when seeking to attain any special object. When at rest the left arm appeared to be retained awkwardly or with difficulty at the left side, with the fingers of the hand extended, or the palmar surface of the fingers pressed firmly against the hip. On moving the arm for any definite purpose the fingers at once became abducted from the middle line, from probable spasm of the dorsal interossei, and the palm of the hand would be drawn inwards, so as to form an arch with the lower part of the forearm from spasm of the flexors. On being told to touch his nose, e. g. with the forefinger, the fingers at once become abducted from the median line, and tend to close inward on the palm; the wrist becomes rounded; the palm with the lower part of the forearm becomes arched; and the hand, spasmodically moving, is first placed on the chest, with the view of pressing the fingers firmly against the body to prevent their closing: the whole hand is then at length sprawled over the face in a vain endeavour to accomplish the task imposed. With the eyes shut, all these movements become more strained, complex, and difficult. On examining the left foot it is clear that it is similarly affected to the hand, and the normal antagonising force between the flexors and extensors is lost. On standing erect he rests the weight of his body on his right foot, and bending over slightly to that side advances his left leg straight outwards from the hip, the knee being unbent, the os calcis drawn up, and the toes

A Case of Athetotis.

DR. LEWIS SHAPTER.

extended with their tips presented to the floor. The condition simulates talipes equinus, with dislocation of the hip into the thyroid cavity. By a strong effort of the will he can restore the balance in muscular action, and the sole of the foot is thus brought flat upon the ground, but this is only for a time, until the opposing muscles are wearied by their overwork.

Beyond this spastic condition of the flexor muscles there appears to be no disorder indicative of nerve lesion ; sensibility to touch, pain, heat, and cold appear normal ; there is neither numbness nor pain ; galvanism of the flexors and extensors affords no reliable results ; but the arm and leg are perhaps somewhat wasted in certain parts, such, e. g., as the calf of the left leg. The abnormal muscular action, it is most important to observe, was continuous rather than jerking, and rapidly recovering or spasmodic. The movements resulting from the tonic muscular spasm, might, indeed, be described as slow in attaining their object, deliberate, and forcible.

The history of the case, according to the account of the mother, was as follows :—When three months old the child was vaccinated on the *left* arm, after which an abscess formed on the outer side of the same arm, which broke and subsequently healed. About this time it was noticed that his hand was constantly moving in a peculiar manner, and when he began to walk he walked on the tips of the toes of the left foot. These symptoms have since increased. The child was always weakly, and the three brothers and three sisters are also delicate ; but, as far as can be ascertained, there is no defined disease in any of them.

The treatment consisted in the use of the bromide, of conium, and of the extractum physostigmatis ; both of which latter drugs were pushed to the production of physiological effects. On one occasion he was placed under chloroform, with the view of observing how much of the acquired distortion depended upon muscular spasm, and with the further view of getting the foot into such a position as to render a boot adaptable. As results, spasm was temporarily relaxed, deformity removed, and a boot adapted ; but this latter, after some days, was obliged to be again removed. Special treatment directed to the existence of spasm was, on the whole, unsuccessful ; but under tonics and attention to general nutrition some benefit was derived.

Under the name of Athetosis (Αθετος, without fixed position) Dr. Hammond first described an affection which is mainly characterised by an inability to retain the fingers and toes in any position in which they may be placed, and by their continual motion. Dr. Hammond illustrated the disease by two cases, one an intemperate epileptic who after an attack

of delirium tremens, followed by unconsciousness for six weeks, complained of numbness in the whole of the right upper extremity and in the toes of the same side, severe pain in these parts, and complex movements, which were to some extent under the control of the will, especially when this was strongly excited and assisted by the eyesight: coincidently with the development of this condition the memory became impaired, and the intellect manifestly weakened. The second case was also an epileptic, who, after an attack of aphasia, developed numbness on the affected side, incoordination and loss of voluntary muscular motion of the right forearm and leg, loss of antagonising force between the flexors and extensors both of the fingers and toes, and slow moving pains from the hand and foot up to the body. Both cases, therefore, came on with epileptic paroxysms; both were characterised by complex movements of the fingers and toes, with a tendency to distortion; both were also associated with similar head symptoms, tremulousness of the tongue, numbness on the affected side, and pains in the spasmodically affected muscles; but in neither case was there any paralysis. Hammond's view as to the pathology of the disease was purely hypothetical; but he associated the disorder with the intra-cranial ganglia and the upper part of the spinal cord, and named the corpus striatum as one probable seat of the morbid process. Since Dr. Hammond described such a special form of unilateral motor disturbance, under the name of Athetosis, in 1871, other illustrative cases have, from time to time, been recorded by Dr. Gowers, Professor Gairdner, Charcot, Dr. Munro, Dr. Claye Shaw, Dr. Sydney Ringer, and Dr. Clifford Allbutt. Some few of these recorded cases have involved general bilateral irregularity of movement, whilst others have been post-hemiplegic and unilateral; but all appear to have been connected with, or preceded by, other disease of the nervous system, and thereby afford evidence that the pathological change (whatever it might be) causing the disordered movement has been secondary to, or dependent on, a permanent lesion of the higher encephalic centres.

In some few cases pathological research has come to our aid. Charcot found in three cases of post-hemiplegic irregular choreoid spasm occurring on movement, lesions involving the posterior extremity of the optic thalamus, the posterior part of the caudate nucleus, and the most posterior part of the corona radiata; and in these posterior fibres of the corona radiata he assumes there is a special motor function, alteration in which causes choreoid spasm. Dr. Gowers ('Medico-Chir. Transactions,' vol. lix.) reports a case of post-hemiplegic incoordination affecting the arm

only without spontaneous mobile spasm: the brain revealed but one lesion, a cicatricial induration of the optic thalamus extending across its centre beneath the upper surface, but not involving the ascending white fibres from the crus. Dr. Sydney Ringer ('Practitioner,' September 1879) in a case of post-hemiplegic athetosis, found the disease on examination to be limited to the optic thalamus, corpus striatum, and the parts just external. The corpus striatum was the part which suffered most, but the left optic thalamus was also wasted. Dr. Ringer remarks that it is obvious that the cavity in the brain could not be the immediate cause of the incoordinate and involuntary movement of athetosis, for this breach of continuity could only cause paralysis. "The athetosis, therefore, must be attributed to the damaged tissues in the neighbourhood of this cavity, and must, in this case, be due to the damage in the optic thalamus or the corpus striatum or both." It is beyond my present purpose to discuss how far the special phenomena of athetosis are to be dissociated from the commoner characteristics of fixed and mobile spasm so often occurring in partly paralysed limbs, and consequently I may be debarred from considering how far atrophy and degeneration of the basal ganglia may be looked to as the sources which give rise to the phenomena peculiar to post-hemiplegic athetosis. Considering the observations of Crichton-Browne and Meynert on the optic thalamus as a reflex centre, we can with Dr. Gowers readily understand how lesion of this organ alone may account for the disease. My desire rather is to look apart from the basal ganglia for a point from which to ground an explanation of the causation of the phenomena peculiar to athetosis as a functional disorder.

According to the observations of Tcheschechin, the centres of innervation of the movements of locomotion are found at the protuberance, and as high up as the cerebral peduncles, and these centres are in communication with the different encephalic centres that are attached to the protuberance by the middle cerebellar peduncles and cerebral peduncles. Lesions of these centres, or peduncles, occasion a disturbance in the co-ordination of movements, and also unilateral lesions to the peculiar movements of rotation, such as whirling, motion on a pivot, rolling, or somersaults. Although, therefore, we cannot physiologically regard the protuberance as the seat of transition between the spinal cord and the higher perceptive or volitional cerebral centres, we can at least accord to a "centre" which is one of the principal seats of the reception of sensations, a function presiding over the movements of locomotion, and liable therefore as a "centre," especially in the young with growing nerve elements, to receive partial or

permanent damage or alteration of function from injury or even shock, direct or indirect, or reflex.

The case of athetosis under consideration is one unassociated with other nerve lesion either past or present, and presumably it is dependent on atrophic changes in nerve elements of spinal centres, and primarily due to shock indirect or reflex.

The clinical features characteristic of the disease are briefly (1) tonic, slow, more or less designed, and perfected muscular spasm, especially of the flexors; (2) motor incoordination; (3) distortion, the result of unopposed muscular contraction; (4) a want of controlling volitional power, whether such want is really dependent upon a loss of power, or only upon a local ataxia of the agent to be controlled.

Now the point naturally at issue to every clinical investigator is the cause of the incoordinate movement; or, in other words, is this incoordination a result of an affection of the co-ordinating spinal centre, or is it merely a mechanical result of flexor spasm which would necessarily give rise to an awkwardness and irregularity of movement?

In locomotor ataxy we have incoordination of movement as a pathognomonic feature, and although this incoordination is, in the majority of cases, intimately connected with spinal lesion, preventing conductivity of motor force, it is not necessarily dependent upon it. As Trousseau observes, it would be wrong to regard the disease as dependent on such conditions as the palpable lesions found, since they are only a consequence and an effect. Incoordination of movement, indeed, when it exists as a purely functional disorder in a class of cases free from any other implication of nervous function, speaks against the *necessary* dependency of co-ordination on the evolution of force from nerve centres. An example of this class of case I ventured to publish, in 1872 ('Med. Times and Gazette'), and I then drew attention to the fact that incoordination, as occurring without the supposed necessary lesion involved in posterior spinal sclerosis, could not be regarded as necessarily dependent upon it; but that we had rather to view, with Dr. Lockhart Clarke, a disorder of that physiological state (viz. tonicity) of the muscles, which, as dependent on reflex action, maintains the constant state of partial contraction, and keeps the antagonistic muscles in equilibrium or static tension. Incoordination of movement may then, clinically and physiologically, be a local disorder in the antagonising muscles themselves, and is the result of a loss of the physiological state of tonicity. In posterior spinal sclerosis, again, there is found a sufficient lesion, not only to interrupt the ordinary cycle of events and to disturb the necessary reflex act upon which co-ordination is dependent;

but also to produce an irritation to react upon the cord as a centre, and so occasion an evolution of motor force upon which harmony of action must be directly due. Incoordination then being, from the nature of the case, a disorder of motor force, and the cord being a generator as well as a transmitter of nervous force, we find in posterior spinal sclerosis motor action, irregularly distributed through the proper nerve channels from a nerve centre, and connected with a morbid condition of the sensory system, or that part which goes to complete the cycle necessary for the production of a reflex act. In athetosis we have only to go a step further, for we have to deal with unilateral incoordination of movement, presumably the result of disorder of a special incoordinating centre, encephalic in location. It follows also that in all cases of post-hemiplegic athetosis there must necessarily be a continuity of function between the motor path and the grey matter of the centres, and it must be assumed that the lesion causing the associative hemiplegia is only partial as regards the grey matter, and consequently permits of that "central irradiation of voluntary impulse" insisted upon by Nothnagel.

Finally, we are asked by many investigators to see a connection between athetosis and chorea, paralysis agitans, writer's cramp, spastic paraplegia, &c. Hemichorea is thought to bear the closest analogy, but if the pathognomonic feature of athetosis is incoordination of movement, and the pathological element disorder of the co-ordinating centre at the protuberance, the disease is sufficiently well-defined to render comparisons of minor clinical details dangerous and inconvenient. Chorea has its own clinical landmarks. In the so-called "insanity of the muscles," in the morbid restlessness, degree of variety, and combination of the involuntary movements; in the rapidly recurring and alternating flexion and extension, pronation and supination, abduction and adduction, occurring in the upper and lower extremities; and finally, in the "dreams of movement" which may occur during sleep, we have surely clinical characteristics that need not be confused or even dwelt upon. In athetosis we find little or nothing in common with chorea; the incoordination of movement, the flexor spasm leading to distortion, and the slow tonic designed character of the spasm, are phenomena distinctive and pathognomonic. It has been with the hope of rendering these signs more generally accepted, that I have thought a case of "functional" athetosis, uncomplicated with any other nerve disorder, worthy of consideration.

CASE OF CONGENITAL ABSENCE OF THE CORPUS CALLOSUM.

BY A. R. URQUHART, M.D.

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THE following is a *résumé* of the bodily and mental characteristics during life, and the post-mortem appearances of a case that occurred under the care of Dr. Parsey, Warwick County Asylum:—

E. E. was for seven years an inmate of the Idiot department of that Asylum. She died of empyema. During life she gave no occasion for any note of interest in the case-books—being regarded as an imbecile with certain vicious propensities rendering it necessary to place her under proper care and control. The rare cerebral defect was only discovered on the post-mortem examination; and thereupon these notes were compiled from the recollections of medical officers and staff. I believe they may be accepted as accurate, though to so great an extent negative in character.

I. *Bodily Characteristics.*—The general appearance of E. E. when in repose called for no remark. She was of middle height, well nourished, and well developed. Expression somewhat fatuous, but not devoid of intelligence.

The shape of head was irregular and globular, taken in the manner recommended by Mr. Crochley Clapham.

The vital functions were duly performed, and she was healthy and vigorous till the fatal illness.

The most marked abnormality was a certain want of muscular co-ordination. This, however, did not obtain to such an extent as to make her the object of general remark among her fellow-patients. She fed herself in the dining-hall with knife and fork in a slightly awkward manner. She could pick up a pin. She was taught sewing, and got as far as hemming dusters, but the result was very indifferent. Indeed, the nurse described it as the "worst sewing she had ever seen." When thus employed she would prick her fingers, and turn out

"childish" work—very irregular in size of stitch and line. Her hands were small, the fingers round and stumpy.

The want of co-ordinating power was most marked in her gait. She took regular exercise round the grounds, and always walked with a gliding motion, never lifting her feet. In the airing-court she was generally to be found moving close by the wall—evidently from preference. She would fall on the slightest push, even if standing still and expecting an attack, so precarious was her balance.

All her motions had a tendency to become automatic. When engaged in scrubbing the floor she wet her garments, and would continue to work at the same spot till moved by the nurse. She used a duster in the same mechanical way. These automatic movements were very noticeable when she scratched her head.

There is unfortunately no accurate record of the state of her special senses. They had been so far normal as to have attracted no notice.

The tone of voice was gruff and peculiar. The nurse in charge stated that she never heard one like it. The only sound she could liken it to was the bark of a dog—not shrill, but thick and sudden.

For a long time after admission she never spoke, till one day the nurse heard a strange and curious voice in the court, and found that it was E. E. scolding. Indeed, her powers of language were seldom exerted save under the influence of rage, when she would swear freely, using many different oaths. She seemed to collect her faculties during a short preliminary pause, and then ran the words of her brief sentences into one another, bringing them out "all of a sudden." This rendered it difficult to make out what she said.

II. *Mental Manifestations.*—E. E. was not possessed of a high degree of intelligence.

She was timid, and stood in awe of her stronger neighbours. If observed working mischief by the nurse, she would desist at once—at times refusing to speak when questioned on the point, and at times in angry tones denying her fault, and blaming another patient by name.

She was occasionally destructive and mischievous, gave much trouble by constant destruction of boots, etc., and by scratching the painted walls and doors.

She constantly pinched and thumped other patients in a very sly way, and was both spiteful and revengeful. Towards the close of her life these traits became more marked, and for that reason she spoke more. Two patients and the night nurse she seemed to regard as special enemies. One of these patients was weakly and inoffensive, the other aggressive and

powerful. It was only when there was but little risk of discovery that she meddled with the latter. So troublesome was she in this respect, that when the nurse found the "ward up in arms, harmless patients and all," she generally found that E. E. was the cause of the tumult.

She gave the night nurse all the trouble she could by wetting her bed; and, if possible, by changing her own fouled linen for the dry clothing of a near neighbour in the dormitory. She was evidently uncomfortable when wet, and never so misbehaved in the daytime. Her general arrangement for the night was to place the macintosh sheet below the mattress.

So troublesome was she in the dining-hall that a nurse kept close beside her to prevent these tricks. The most common was her inveterate habit of stealing—not from hunger, but apparently for the mere love of mischief. She would sip a little out of her own cup and exchange it for the full one of a helpless neighbour; or as she passed along would rapidly throw the slices of bread underneath the table, with no intention of eating it.

She did not give signs of pleasure readily, being singularly apathetic; but she showed considerable fondness for children and others. At night she carried certain helpless little ones upstairs to the dormitory, and had an evident preference for them. She, however, performed this duty in a very clumsy fashion, as might be expected, getting upstairs crabwise and slowly.

The nurse in charge of her ward was a special favourite. She would promise this nurse to keep her bed clean; but soon forgot her promise in telling the night attendant that she meant to "serve her out" by misbehaving.

She exhibited no sign of jealousy.

The faculty of attention was not strongly marked. While in the Asylum school she would intently stare at her teacher for a time, but nearly always had to be turned out on account of her mischievous propensities, e.g., for kicking the others underneath the table. She never showed any sign of having learnt anything, though she had been tried with object lessons, shapes, numbers, colours, and reading.

She had little or no idea of time, and required to be called to meals. She would (but rarely) tell the head attendant of the department that "the doctor was coming." There was a certain amount of reasoning and reflective power. Ideation, however, was very slow; she always paused before speaking, and during her illness she took little, if any, interest in her treatment, and was awkward and tardy in obeying instructions.

Her habits were untidy and filthy.

She was most acquisitive. Did she want anything (say a comb or brush), she would not return it to its place; but quickly and secretly hide it away. But she seemed to make no use of accumulated finery or rubbish. For years she carried about twopence-halfpenny in coppers in her hand, till they became green and worn. One day, when visited by a friend, she changed them for clean coins, but never evinced any desire or idea of spending them.

In conclusion, we find that the chief bodily want was a deficiency in co-ordinating power over the muscles; while the mental faculties were, generally speaking, developed to a slight extent. The higher faculties were either entirely absent or present in a slight degree. Attention, imitation, ideation, the moral sense, were extremely limited in scope; and education seemed to fail in developing them.

III. *Post-mortem Examination: Head.*—Calvarium very thin, extremely irregular in shape, shortened antero-posteriorly, and nearly circular. The greatest diameter was from the right frontal to the left occipital region. The measurements of the interior of the skull were: length, 6 inches; breadth, 5 inches; depth from vertex to posterior clinoid processes, $3\frac{1}{4}$ inches; circumference, $17\frac{1}{2}$ inches. The right side of the skull was flattened posteriorly, and bulged slightly anteriorly, so that the hemisphere of that was, as it were, somewhat pushed forward.

Dura mater non-adherent. Frontal lobes much shortened, orbital depression very marked. Convolutions small and simple, especially in frontal and occipital lobes. Corpus callosum represented by a rudimentary ridge on either hemisphere. Gyrus fornicatus convolution absent, numerous radiating convolutions taking its place.

On removing the brain, which was soft and flabby, the hemispheres at once fell apart, disclosing the absence of the corpus callosum, together with the fornix and septum lucidum. A thin pellucid extension of pia mater served to connect the hemispheres.

The small size of the cerebral hemispheres, the simple character of the convolutions, and the absence of the corpus callosum, were the most remarkable features. In other respects the brain was apparently normal.

Remarks.—Cases of this abnormality have been recorded by Paget, Sully, Reil, Langdon Downe and Christie.

In all (as far as I have been able to ascertain) the mental faculties were defective. But in cases where the abnormality was incomplete, where there was only a partial arrest of

development of the corpus callosum, fornix and septum lucidum, the intellectual functions were not so much impaired.

The case described by Sir James Paget was an example of this pathological condition, and had been a domestic servant, whose mental aberration was confined to a want of forethought and an abrupt flighty manner. She had a slender education, a good memory, was trusty and competent in her sphere of labour, and bore a good moral character.

The other cases showed very low degree of mental development, with very limited command of language. The case above recorded presents a comparatively high standard, notwithstanding the extensive absence of the commissural system.

We may conclude that these structures are necessary for a high degree of mental evolution, that their partial absence is attended by slight intellectual defects, while their total loss causes varying degrees of idiocy and imbecility. It is impossible to affirm that speech is affected by this want more than any other intellectual faculty.

Abstracts of British and Foreign Journals.

The Central Sulci in the Carnivora.—Professor Pausch, of Kiel, in the course of a monograph upon the Morphology of the Mammalian Brain, describes the sulci in the cerebrum of the Carnivora (Beiträge zur Morphologie des Grosshirns der Säugethiere. I. Die Furchen am Grosshirn der Carnivoren. *Morphologisches Jahrbuch*, Bd. v. ss. 193–239, Taf. xiv. u. xv.). The first portion of his paper is devoted to the Canine, Feline, and Ursine families, and is illustrated by two double plates of fifty-five figures.

I. The Dog group. Canina.—The brains of the fox and of the dog, the latter in various stages of development, are described and figured. The author concludes that the brains of the fox, wolf, and dog have the same type in their sulci; and that, further, when this type shows itself in the simplest form in any species it is also subject to the most trifling individual variations, and *vice versâ*. Thus, all fox brains display almost precisely the same sulci, while in the dog great variations may be seen, the two halves even of the same brain displaying maybe seemingly fundamental differences. It is true that the probable clue to this should be sought for in the numerous varieties or races into which the dog, as the oldest and most widely distributed domestic animal, has become separated.

II. The Cat group. Felina.—The brain of the cat in its various stages, and that of the lion, tiger, leopard, panther, and hyæna. Taking the brain as a basis of comparison, the feline tribe is well marked out as a natural group according to Professor Pausch; for there, while the sulci show on one side or surface agreement with those of the canine brain, on the other side they display characteristic variations therefrom.

III. The Bear group. Ursina.—The brain of the marten and weasel, the otter, the coati and raccoon, the bear, and the mungoose and genette, are described and figured. In the ursine group it is concluded that there is no such agreement in the typical features

of the brain as exists in the two preceding divisions, and that accordingly three sub-groups may be formed, viz.:—

a. That including the musteline and ursine families. Through the coati a transition may be made to the next sub-group.

b. That comprising the lutrine family, including the otter and raccoon.

c. The sub-group formed by the viverrine family, to which may be added the genette and mungoose.

From a study of the brains of the animals above mentioned, Professor Pausch has constructed a diagram of the sulci and gyri of the typical Carnivora brain.

The Brain of the Hippopotamus.—In a recent number of the Transactions of the Zoological Society (vol. xi., 1880, p. 11) there is a description, accompanied by five figures, by the late Professor Garrod, of the brain of an adult specimen, about thirteen years old, from the Upper Nile, of *Hippopotamus amphibius.* Professor Gratiolet had previously described the brain of the same species in a well-known monograph on its anatomy ('Recherches sur l'Anatomie de l'Hippopotame,' Paris, 1867); and Professor Macalister has given an outline sketch of the same organ in *H. Liberiensis*, *Proc. Roy. Irish Acad.* vol. i. ser. 2, pl. xxviii.

The weight of the brain immediately after removal was 1 lb. 7 oz. Professor Garrod adopts the nomenclature used by Dr. Krueg in a recent monograph on the Brain in the Ungulata (*Zeitsch. für wissensch. Zool.*, 1878, ss. 297–344).[1] The brain is not richly convoluted, being about as much so as that of the genus *Bos*, and decidedly less so than that of *Camelopardalis giraffa* or the Camelidæ. The brain of *Ceratorhinus Sumatrensis*, a much smaller Rhinoceros (see *Trans. Zool. Soc.* vol. x. pl. lxx. p. 411), is much richer in convolutions. The arrangement of the convolutions on the two sides of the brain which is the subject of this note differs so much that, from a study of one or the other singly, very different results might be arrived at. This is evidently due to the considerable development on the right side of bridging convolutions, the great number of which in the brain of the hippopotamus is particularly remarked by Gratiolet, who, in reference to the "middle series" of convolutions, remarks: "Il acquiert une importance exceptionnelle, et si son existence est au premier abord dissimulée, cela tient à la grande quantité de plis de passages verticaux qui lient cet étage supérieur à l'étage inférieur proprement dit." On the left side these bridging con-

[1] Abstract in 'BRAIN,' Part IV., p. 379.

volutions do not exist. The Sylvian fissure is insignificant in size, the fissura rhinalis being continuous with it before and behind. As already remarked by Gratiolet, the optic and olfactory lobes are small, and the corpora quadrigemina not greatly developed. "Looked at generally," concludes Professor Garrod, "the brain of the hippopotamus is evidently very different from that of the genus *Sus* and its nearest allies. In the great breadth of and complicatedness of what, in my paper on the brain of the Sumatran Rhinoceros (*Trans. Zool. Soc.* vol. x. p. 411),[1] I term the middle oblique convolution (that between the lateral and supra-sylvian fissures), it most resembles the Camel and the Giraffe, from the form of which it strikingly differs in the much less 'pronation,' as Dr. Krueg terms it, of the hemisphere. On the whole, it stands very much by itself."

The Brain of the Bush Dog. (*Icticyon venaticus.*)—In the last number of the Proceedings of the Zoological Society (part i., 1880, p. 73) Professor Flower in the course of a paper on the anatomy of the Bush Dog (*Icticyon venaticus*) describes and figures the brain of this animal. This organ is stated to be characteristically canine, except that on the left side the gyrus immediately surrounding the Sylvian fissure is not marked off by a complete sulcus at the upper curved part from the one above it. It thus almost reproduces the condition met with in the Felidæ, from which form, according to the view of the late Professor Garrod, the canine brain has been derived by complete division of the lower or external gyrus into an outer and inner segment (Notes on the visceral anatomy of *Lycaon pictus. Proc. Zool. Soc.* 1878, p. 377.)

"Although I have no doubt," concludes Professor Flower, "after examining a larger number of specimens than were available when attempting a classification, and comprised of the cerebral convolutions of the different groups of the Carnivora, that the fourth—counting from the middle line—or inferior gyrus of the dog, is represented by the outer or inferior portion of the third (counting in the same way) of the other Carnivora, and is in many Æluroids already partially marked off by an interrupted sulcus, I am not prepared on that account to accept the conclusion that the dog is a further modification of the highly specialised Æluroid type. Very little else in the structure or the palæontological history of the dog indicates that it has passed through a feline stage in its development, and its more complex brain may have been cortical quite independently from a primitive form."

J. C. Galton.

[1] Abstract in 'Brain,' Part III., p. 418.

Exner on Cerebral Localisation.—At the meeting of the Academy of Sciences at Vienna, June 17, 1880, Exner gave the following as the results of his investigations on the functions of the cerebral cortex. He had limited his examination to cases of purely cortical lesions—169 in number—recorded in literature. The results, which will be published more fully at a later date, are, that in the cortex there are areas which are in relation with certain functions in a limited, and others in a more general sense.

In general it is lesions of the two central convolutions which paralyse or impair volitional movements on the opposite side. But in addition to these the greater portion of the convex surface of the hemisphere must also be reckoned in the motor zone, though of less significance.

In the motor zone there are areas corresponding to certain muscular actions which may be termed absolute, when lesion of these invariably causes affection of these movements; relative only, when this is not constant. Only the muscles of the extremities and the right side of the face have an absolute cortical area; the others, only relative cortical regions.

All the motor areas have a greater signification, and, in a certain sense, also greater extension, in the left hemisphere than in the right. They are not sharply demarcated, but cease gradually. Many muscles, those which are usually associated together, have areas in both hemispheres. Those muscles which usually act together, whatever their peripheral nervous supply, are intimately related together in the cortex.

As regards speech, the temporal convolutions are not less important than the frontal or the insula. Lesions of the temporal lobes, especially affecting the middle temporal convolution, cause the affection of speech termed "word deafness."

As to the sensory areas, it appears that as the left hemisphere is especially motor, so is the right especially sensory. The tactile areas of the extremities correspond with their motor areas. They are not separate, but the centres are conjoint sensory and motor.

The centre of vision is in the first occipital convolution, and, like the motor areas, shades gradually into the surrounding cortex. Lesions of this centre cause ocular hallucinations, or disorders of visual perception, or hemianopy. Each sensory centre seems to be in relation with both sides of the body.

H. Obersteiner.

Brachial Monoplegia.—Déjerine (*Prog. Méd.*, August 14, 1880) records a case of right brachial monoplegia, combined with contracture and tremor, and also with anæsthesia. The mobility of the face and leg was unimpaired, but the sensibility of the leg was greatly diminished, though less so than in the arm. The special senses were unaffected.

After death a tubercle of the size of a nut was found in the optic thalamus compressing the internal capsule towards its posterior aspect.

He considers this case especially interesting as showing that monoplegia may occur apart from purely cortical disease, indicating that the medullary fasciculi for the arm and leg remain distinct, and are capable of being individually and separately affected.

[The case however, complicated as it was with anæsthesia and tremor, is quite unlike monoplegia depending on cortical disease.]

Pitres on Cerebral Localisation.—Pitres (*Prog. Méd.*, Aug. 7, 1880) reports several cases which have recently come under his observation illustrative of the effects of cerebral lesions, according to their position within or without the motor region.

The two first relate to lesions of the sphenoidal, parietal and occipital lobes, without motor symptoms. The third was a case of lesion of the nucleus caudatus of the corpus striatum, also without motor symptoms, showing that when the lesions do not also involve the internal capsule paralysis does not ensue, and indicating, therefore, the necessity of accurately discriminating between lesions of the grey matter and the capsular fibres. The fourth case was one of extensive destruction of the motor zone and third frontal, causing persistent hemiplegia and aphasia. The fifth was one of right hemiplegia and hemianæsthesia, the lesion being in the internal capsule and foot of the corona radiata. The aphasia was explained by the extension of the lesion into the inferior pediculo-frontal fasciculus, thus cutting the medullary connections of the third left frontal, though the cortical substance was not destroyed. The sixth case was one of brachial monoplegia depending on limited lesion of the centrum ovale in the middle pediculo-frontal fasciculus. Secondary degeneration was demonstrated also in the spinal cord in two of the cases of lesion of the motor zone, and its absence demonstrated in one where a lesion of old-standing existed outside the motor zone—inferior parietal and sphenoidal lobes.

Kronecker and Nicolaides on Irritation of the Vaso-motor Centres by Summation of Electrical Stimuli.—In a paper on this subject (*Verhandl. der Physiolog. Gesellsch. zu Berlin*, No. 17, 1880) the authors arrive at the following results:—

Single induction-shocks applied to the vaso-motor centres of the medulla or below it, after removal of the cerebral hemispheres, have no influence on the blood-pressure, or only slight, with currents which are so strong that practically each shock is a tetanising one.

Moderately strong stimuli are only active—by summation—when they occur at least two or three per second.

These slowly repeated stimuli gain in effect when their intensity is increased, but it is never possible with such form of stimulation to cause the maximum effect such as is produced by moderately strong stimuli of greater frequency.

If the intensity remains constant and the frequency increased, the effect becomes more marked. The effect is not increased when the frequency of the stimulation exceeds 20–25 shocks per second.

This maximum of vascular contraction, which varies in different animals, can also be produced by strong stimuli of moderate frequency (10–12 per second), and also by moderate stimuli of maximum frequency (20–25 per second).

The maximum of vascular contraction in consequence of strong but less frequent stimuli is not reached so soon as by weaker and more frequent stimulation.

After the irritation of the vaso-motor centres ceases, the blood-pressure sinks gradually, just as in Ludwig and Baxt's experiments on the accelerator nerves of the heart, while the inhibitory action of the vagus very rapidly disappears. Hence, with vagi intact, irritation of the medulla oblongata at first only inhibits the heart, the blood-pressure first beginning to increase when the vagal irritation has subsided.

These laws apply also in the case of irritation of the peripheral ends of the divided splanchnics. Here, however, the intensity of the effect varies, as is known, with the animal.

The complete analogy of the above results with those obtained by summation of cutaneous electrical stimulation, allows the conclusions to be drawn—

1. That vascular constriction is caused by irritation of the medulla oblongata, spinal cord, and also of the splanchnics in a reflex manner.

2. That just as the motor cells of the spinal cord, under whatever form of tetanisation, vibrate with a constant frequency which

corresponds to the normal muscle sound, so the vaso-motor centres have a tone or vibration of their own—corresponding nearly to that of the muscles.

Marckwald and Kronecker on the Respiratory Centres.—Marckwald and Kronecker (*Verhandl. d. Physiolog. Gesellsch. zu Berlin*, August 9, 1880) publish the results of a research with reference to the influence of electrical irritation of the vagi on the respiratory movements. They find that animals (rabbits) whose medulla is severed by a transverse incision at the level of the *striæ medullares* of the fourth ventricle, continue to breathe for several hours in a fairly normal manner. In the medulla there are two distinct excitable centres, one inspiratory, and a weak expiratory, whose alternating action is the cause of rhythmical respiration. These centres are stimulated to action by the vagi, which are, however, supplemented by other centripetal nerves—such as the cutaneous nerves. This is shown by several facts. Section of the vagi in the neck puts a stop to regular respiration. By irritation of the proximal ends of both vagi, both inspiration and expiration can be excited, more especially inspiration. By rhythmic electrical irritation of the proximal ends of the vagi, reflex artificial respiration of a fairly normal type can be excited. This can be effected without the vagi, but less easily, by a powerful cutaneous irritation.

The inspiratory and expiratory centres seem to stand in a similar relation to each other as the vagus and accelerans, or as the vagus nucleus and the vaso-motor centre. A simple electrical stimulus of the vagus causes a respiratory movement only in combination with other stimuli. Continued or periodical tetanising irritation causes rhythmical respiration. Even during apnœa tetanising stimulation of the vagus causes incomplete respiratory movements. Single electrical stimuli do not produce this effect. These reflex respiratory movements are shown more especially in the diaphragm; but the other muscles of respiration are also capable of being similarly acted on, though the stimuli must then be stronger and more frequent.

Ferrier and Yeo on the Cerebral Visual Centres.—At the meeting of the British Medical Association at Cambridge, in August, Professors Ferrier and Gerald F. Yeo read a paper before the Physiological Section, in which they gave a preliminary account of

their recent investigation of the visual centres in monkeys. The operations were conducted under the Listerian antiseptic method, by which lesions can be established in the brain with a total absence of encephalitis or other secondary changes, which almost invariably accompany ordinary operative methods, and more or less complicate the results. After alluding to the previous views of Ferrier, Munk, Luciani and Tamburini, they gave the following as the results of their research :—

1. As to lesions of the occipital lobes.

They found that neither as the result of destruction nor bodily removal of one or both occipital lobes, provided the lesions did not trench on the parieto-occipital fissure, was there any discernible disturbance of vision, or other bodily or mental functions.

2. As to lesions of the angular gyri.

After complete destruction of one angular gyrus there is temporary loss of vision in the opposite eye, lasting only a few hours. The common sensibility of the eye remains acute, and there is no ptosis or other muscular paralysis.

The restoration of vision is not due to the integrity of the other angular gyrus alone. In regard to this point the results differ, according as the angular gyri are destroyed simultaneously in both hemispheres, or the one angular gyrus several weeks after the other, after complete recovery from the first operation. In the latter case vision is either not markedly impaired, or, if so, only in a very transient manner. Within a few hours an animal deprived of both angular gyri *successively* sees, to all appearance, perfectly well with both eyes. Even though the portions of the occipital lobes immediately adjoining the parieto-occipital fissures are also removed, vision is not perceptibly impaired. If, however, both angular gyri are destroyed *simultaneously*, total blindness ensues in both eyes. But this does not last more than three days. After this period the animal gives evidences of vision, though impairment of vision is distinctly indicated as long as a month afterwards.

It follows, therefore, from these facts that without its occipital lobes a monkey *continues* to see; and that without its angular gyri it can nevertheless *regain* its sight.

3. As to destruction of the angular gyrus and occipital lobe together on one side.

This lesion causes very evident loss of vision in *both* eyes towards the side opposite the lesion. Whether the hemiopia is quite symmetrical they do not decide positively, but they regard the bilateral affection to be unquestionable. This hemiopia is, however, also

merely temporary. At the end of a week there is distinct indication of vision to both sides.

Remarkable results were obtained by what may be described as diagonal lesions of the visual centres.

In an animal whose left angular gyrus had been destroyed (recovery), the right angular gyrus and occipital lobe were destroyed. As the result of this there was left hemiopia, from which, however, the animal recovered completely within a fortnight. This animal, therefore, had only one occipital lobe.

In another animal which had had both occipital lobes previously removed without symptoms, the left angular gyrus was destroyed. The first effect of this was transient loss, followed by indistinctness of vision in the right eye. Within twenty-four hours the animal was again perfectly well, and in full possession of its visual faculties. In this case one angular gyrus alone remained.

From these two cases, therefore, it appears that one occipital lobe or one angular gyrus can suffice for vision with both eyes.

4. As to destruction of both angular gyri and both occipital lobes.

The result of this lesion is total, and to all appearance permanent, blindness in both eyes, without any impairment whatsoever of the other sensory faculties or motor powers. An animal with this lesion had, after months, gradually acquired some considerable confidence in its movements; but though in some of its actions it sometimes appeared as if it saw, yet the authors had never seen any indubitable indication of vision, or anything not more easily explicable by the keenness of its other faculties.

They regard this result as one of the most conclusive demonstrations of the localisation of function in distinct cerebral regions.

Debove and Gombault on the Decussation of the Sensory Tracts in the Medulla Oblongata.—In a note on this subject (*Archives de Neurologie*, part i. vol. i., July, 1880), Debove and Gombault, after indicating the views of Meynert, &c., state that the sensory tracts fuse more or less completely with the motor tracts. This is rendered evident by the examination of a case of amyotrophic sclerosis in which the medulla was affected. The note is illustrated by figures.

At the lower portion of the decussation the sensory fibres divide into small bundles which penetrate the pyramids in their posterior and external aspect, and then curve round and follow a course parallel to them and mixed up with them.

A little higher the decussating sensory fibres penetrate the motor pyramids in their posterior and middle aspect. A certain number of them fuse with the fibres of the stratum zonale.

At the upper extremity the sensory fibres follow different courses. The most superficial penetrate the pyramids at their external aspect. The most deep apply themselves to the posterior and external aspect, and ascend vertically. Hence it appears that the greater portion of the sensory tracts after decussation become intimately mingled with the motor pyramidal strands.

Debove and Boudet on Ataxic Incoordination.—MM. Debove and Boudet (*Archives de Neurologie*, vol. i. part i.) from an investigation of the muscular system of ataxics support Lockhart Clarke's view that the incoordination of ataxics depends on *inequality* of the muscular tone. An examination of the muscles by the *myophone* (see Boudet, *Soc. de Biologie*, Jan. 1880) indicates considerable differences in the tone of different muscular groups. The habitual diminution of tonicity in the quadriceps cruris accounts for the absence of the "tendon-reflex." In order that this should be manifested a certain tension of the muscles is necessary. Differences are also demonstrable in the latent period between percussion and contraction of different muscles. The inequality of tone serves to account for the ataxic incoordination. It is not the total absence of muscular tonus, such, for instance, as may be induced by action of the posterior roots. This operation in animals causes a peculiar defect in movement, but it is not the same as ataxy.

Though the muscles are unequal in tone, yet the absolute contractile force does not seem impaired, for tonic and atonic muscles, *cæteris paribus*, will contract equally powerfully. The difference is in the degree of stimulation they require. A tonic muscle will reach its maximum contraction sooner than an atonic one. Hence the same volitional stimulation will affect these muscles unequally. The irregularity is most manifest in movements requiring only moderate muscular force, such as standing and walking. The more energetic the movement, the less does the inequality of tone manifest itself. Hence ataxics in all their movements, whether of prehension or locomotion, exhibit a *brusquerie* and exaggeration, which is an attempt to correct the incoordination by a maximum contraction of all the muscles involved.

Gaskell on the Tonicity of the Heart and Blood-vessels.—In the *Journal of Physiology*, vol. iii., No. 1, Gaskell discusses the

influence which alterations in the nutritive condition of the muscular substance of the heart and blood-vessels have on their tonicity. It is commonly supposed that the condition of the heart and the tonicity of the vessels are regulated by purely nervous mechanism, but the experiments he has made on the influence of acids, alkalies, and other substances, seems to indicate that something at least must be ascribed to the nature of the fluids which bathe the tissues. Dilute alkaline solutions have a tonic or constrictive action on the muscular tissue of the heart and arteries; while dilute acid solutions have an opposite or atonic influence. Variations in the alkalinity will affect the tonicity. He supposes that the acidity generated in organs by activity may be influential in causing the dilatation or atonic condition of the blood-vessels which accompanies activity. During rest the tissues are alkaline, and therefore the vessels are in a state of tonicity. In the case of the heart he suggests that the normal beat depends partly upon the maintenance of a due tonic condition of the muscular tissue, which tissue is capable of variation between two opposite extremes of tonicity and atonicity (systole and diastole). He considers various hypotheses as to how this may be brought about, and as to the similar action of digitalis, and certain other cardiac agents. On these the reader is referred to the paper itself. He proposes to discuss at a future date the bearing of his observations on the theory of inhibitory nerves, the nature of recovery of tonicity after removal from the central nervous system, and the action of alkaline medicines and digitalis in the febrile state.

Gombault on Segmentary Peri-axillary Neuritis.—Gombault (*Archives de Neurologie*, vol. i. part i., July, 1880), publishes the first instalment of an investigation of a form of neuritis, designated as above, which in appearance is essentially different from the Wallerian degeneration of nerves. His researches are based mainly on the effects of lead-poisoning in guinea-pigs. In these animals, after administration of lead for several months, changes were induced in the nerves of the characters described, though they did not exhibit any signs of lead paralysis. For a full description reference must be made to the original paper, which is copiously illustrated. The chief points of difference between the segmentary neuritis and the Wallerian degeneration are as follows. In the latter the degeneration is visible simultaneously throughout the whole length of the fibre, and affects the axis cylinder as well as other parts. Regeneration is possible by the development of new

tubes. In the segmentary axillary neuritis, however, the degeneration affects only portions of a nerve, the axis cylinder remains unaffected, and regeneration takes place by the formation of a new sheath of myeline round the intact axis cylinder.

This peri-axillary neuritis also occurs in man, and the author refers to cases of muscular atrophy, amyotrophic lateral sclerosis, and traumatic neuritis, in which the characters of this form of neuritis were distinctly indicated. In man, the Wallerian degeneration is also frequently observed in conjunction with peri-axillary neuritis, and the question will be whether these two forms of parenchymatous neuritis are distinct, or whether they are only different phases of the same morbid process.

Grasset on Locomotor Ataxy and Heart Disease.—In a short and suggestive paper published in the *Montpellier Médical* for June 1880, Grasset brings forward certain facts which tend to establish a causal relationship between locomotor ataxy—especially the painful form—and cardiac lesions. Berger and Rosenbach have already (*Berl. Klin. Wochensch.*, July 7, 1879) published seven cases of the coincidence of locomotor ataxy and aortic incompetency, and think there is some special relationship between the disease and this particular valvular lesion; but Grasset brings forward two cases observed by himself, and cites fifteen others from different authors, in which locomotor ataxy and cardiac valvular disease of various kinds had been noted. There is, therefore, no special relationship between aortic disease and ataxy. The question is whether the two diseases are merely coincidences, or whether there is any causal relationship between them. There certainly does not seem any ground for regarding the ataxy as secondary to the cardiac affection; nor, on the other hand, is there any indication in the cases recorded of the cardiac affection being due to direct action of the diseased cord.

The majority of the cases of tabes complicated with cardiac affection have been remarkable for the intensity and duration of the pains. Hence arises the question whether we may not attribute the cardiac disease to the *painful* character of the malady.

In support of this method of causation Grasset quotes several recent physiological researches—more especially those of Franck, and of Couty and Charpentier—on the influence of irritation of sensory nerves, and of *conscious* pain (emotion, &c.) on the circulation. That such agencies affect the heart—causing stoppage, retardation, quickening, and variations in the vascular tension according to

circumstances—is clearly established by these as well as by many former researches; and that such agencies may actually lead to the establishment of organic cardiac disease is not only possible, but is strongly indicated by many clinical facts.

Most of those who have directed attention to the etiology of cardiac disease—apart from the all-overshadowing rheumatic origin—more especially those observed in connection with gastro-hepatic ailments, have endeavoured to furnish a mechanical explanation secondary to changes in the pulmonary circulation. Grasset, however, has a wholesome distrust of purely mechanical explanations, and looks rather to a direct reflex effect on the heart itself originating from the painful nature of the malady. In this direction, therefore, he looks for an explanation of the probable relationship between ataxy and heart disease.

Tschiriew on Lesions of the Spinal Cord and Skin in a case of Anæsthetic Leprosy.—In the *Archives de Physiologie* (2me série, 1880, p. 614), Tschiriew publishes the results of his microscopical examination of the spinal cord and finger of a leper who died under the care of M. Hillairet in the Hôpital Saint-Louis, in May 1875. The parts had been preserved in a solution of bichromate of ammonia. Nothing abnormal was discoverable in the brain or spinal cord at the post-mortem examination by mere naked-eye inspection.

On microscopical examination of the spinal cord in the *cervical* region, there was no appreciable alteration in the configuration of the white and grey substance. The central canal and its neighbourhood were filled with small rounded granules, colouring intensely with purpurine or hæmatoxyline. In the white and grey substance small, more or less elongated, bodies were scattered, taking on colour also very readily. The medullary veins were congested. In the middle of the cervical region, but only on the left side, there were capillary hæmorrhages occupying the anterior part of the posterior horn.

The anterior horns, with their cells and processes, were normal.

In the posterior horn there were the following changes. The number of the cells was diminished, and they were more or less altered; only a few could be discovered with processes. In general they were reduced to rounded bodies, but retained nucleus and nucleolus. Others had not merely lost their processes but their margins were indistinct, and the protoplasm surrounding the

nucleus reduced to a very thin layer. These cells were sometimes surrounded by lacunæ. There were no vacuolated or pigmented cells (Hayem, Déjerine), nor were any distended (Charcot) cells visible.

The grey substance itself occasionally was without any trace of organisation, being reduced to a more or less opaque, granular mass, taking on colour more intensely than the normal tissue.

In this mass there were small bodies of a rounded form, probably resulting from the atrophied nerve cells.

All the lesions, with the exception of the hæmorrhages, were symmetrical.

The *dorsal* and *lumbar* regions presented similar appearances—the hæmorrhages excepted. There was also alteration of the cells of Clarke's vesicular column, chiefly at the upper and middle regions. The changes were not always equally marked on both sides. A few of the cells of the anterior horns of the dorsal region had fewer and less distinct processes than normal, but those of the lumbar region were perfect.

The *anterior roots* were normal; the *posterior* roots had their sheath a little thickened. The ganglia of the posterior roots had not been preserved.

The *skin* had undergone alteration chiefly in the cutis and subcutaneous tissue. There were infiltrations of small rounded lymphatic or embryonal cells, chiefly in the vascular regions around the glands, and in the adipose tissue. The infiltration seemed to proceed from the small vessels and capillaries, as it was thicker around these than in the neighbourhood of the larger vessels. There was well-marked endarteritis of the vessels.

The *nerves* of the finger were only discoverable by their position, being converted into bundles of connective tissue containing a few small rounded cellules.

The changes observed in the spinal cells Tschiriew regards as being due to *simple degenerative atrophy*, as distinct from pigmentary, vascular, sclerous, &c. As to the grey matter the change seems to be due merely to a destructive parenchymatous process.

What relation there is between the degeneration of the cells and of the grey matter, and the connection between these and the phenomena of anæsthetic leprosy, are all questions which it will be for future research to decide definitively.

Pott's Disease and Progressive Muscular Atrophy.—Proust and Ballet (*Revue Mensuelle*, June 1880) record a case of Pott's

disease in which there was typical muscular atrophy in the thenar eminences and interossei muscles.

On microscopical examination after death there was found sclerosis of the internal radicular zones of the posterior cornua similar to what is seen in locomotor ataxy, atrophy of the anterior horns, and degeneration of the anterior roots of the cervical region, middle and inferior; and lastly, atrophy of the muscular fibres corresponding.

Muscular atrophy is not common in Pott's disease. As to the pathogeny, the authors, after considering the hypothesis of pachymeningitis and ascending or descending degeneration consequent on compression, believe that the condition can best be accounted for as follows. The irritation of the posterior roots by the neoplasia, propagated to the strands of Burdach by the internal root zones, sets up a periradicular inflammation and sclerosis. This propagated to the anterior horns, leads to a degeneration of the multipolar cells and thus to muscular atrophy. They quote some observations by Michaud (*Sur la méningite et la myélite dans le mal vertébral*, Thèse, 1877) in support of myelitis thus capable of being induced. D. FERRIER.

The Pathology and Pathological Anatomy of the Central Nervous System.—Kahler and Pick contribute a lengthy paper on this subject to the *Archiv für Psychiatrie*, vol. x. parts 1 and 2. The cases, especially the histological results, are reported with great fulness and care.

(1.) *Disease of the Posterior and Lateral Columns.*—A man, aged 39, who had often been in hospital suffering from chronic myelitis, died of typhus fever. In the fresh state the cord showed no marked signs of disease, but after hardening the following changes were observed: In the cervical and upper dorsal regions the margins of the cord were converted into a belt of sclerosis. In the lower dorsal and lumbar regions there was a systematic affection of the pyramidal tracts of the lateral columns, in the cervical region the systematic nature of the lesion was less evident owing to the implication of other parts, but the sclerosis was further advanced in the pyramidal tracts than elsewhere. The disease had spread by contiguity to the lateral limiting layers (seitlichen Grenzschicht der grauen Substanz, of Flechsig). The direct cerebellar tracts were sclerosed, and from the fact that the cells of Clarke's columns with which they are connected were abnormally few in number, Kahler and Pick think that the sclerosis of these tracts was not merely an

extension of the disease from neighbouring parts, but was an independent, systematic affection. There was sclerosis of the inner parts of the anterior columns, but whether this was a systematic affection of the pyramidal tracts of the anterior columns or an extension of the marginal sclerosis, is not known. In the upper cervical portion of the cord, the pathological changes in the posterior columns were chiefly limited to Goll's columns, in the lower cervical and upper dorsal portions to the *zones radiculaires post.*, Goll's columns being here almost intact. In the grey matter of the cervical region was a focus of granular degeneration. In a case of secondary descending degeneration, Eisenlohr has described a similar focus in the dorsal region. The two cases go to show that with chronic systematic affections of the white matter may be combined acute affections of the grey matter. As the clinical history is incomplete, the authors leave it undecided whether this was a case of combined systematic disease or a complication of two or more systematic diseases.

(2.) *Compression of the Cord.*—A girl, aged 18, fell on her back and fractured the sixth cervical vertebra. An examination shortly afterwards revealed complete loss of motility and sensibility in the lower extremities; paresis of the arms and paralysis of the bladder. Twelve days afterwards a large bed-sore had formed in the sacral region, and seven weeks afterwards the arms became paralysed. The patient died twelve weeks after the accident. Before death the original bed-sore had almost healed. The cord opposite the 6th cervical vertebra for a space of 1½ ctm. was completely destroyed. Between the thickened and approximated membranes was a small remnant of tissue which was seen to be a network of fine fibres, in the meshes of which were large, flat, slightly granular cells of an epithelial type. Blood-vessels with thickened walls ran in the tissue, and the dilated lymph-channels which surrounded them were filled with cells similar to those just described. There was scarcely a trace of nervous structure. Above and below the point of injury the cord presented the appearances characteristic of myelitis from compression. In some places where the fibres had disappeared from their framework, their place had been taken by endothelial cells. Those portions of the cord that lay next the dura often showed a wide-meshed reticular structure with here and there tube-like spaces. Both meshes and spaces were filled with endothelial cells. Leyden thinks that these cells are analogous to granule-cells, and spring from the neuroglia elements. Kahler and Pick, on the other hand, regard them as growths origi-

nating from the endothelium of the adventitious lymph-spaces. They have to do, perhaps, with a process of regeneration.

For some distance above the place of injury there was ascending degeneration in the direct cerebellar tracts, in Goll's columns, and to a less extent in the adjoining portions of Burdach's columns. In the lower segment of the cord a short tract of secondary degeneration was observable in the posterior columns, and in the same columns was a focus consisting of closely packed endothelial cells and fragments of medulla. A similar focus has been described by Westphal, and it is probably an independent patch of myelitis resulting from the shock of a severe injury. It is allied to the traumatic degeneration of Schiefferdecker.

The inconstancy of the occurrence of ascending degeneration of the direct cerebellar tracts after injury of the lateral columns has often been commented on. Bouchard thought the height of the lesion determined its appearance or non-appearance; when the lesion lies above the mid-dorsal region there is degeneration, when below it there is none. Flechsig attributes the different results to the character or degree of the lesion. Kahler and Pick show by reference to a table of twenty-seven cases of secondary ascending degeneration that neither theory embraces all the cases. But if we remember, first, that the direct cerebellar tract as it ascends from the upper lumbar portion of the cord to the inferior cervical portion, is continually reinforced by fibres from the cells of Clarke's columns, and secondly, that it is only the fibres which are cut off from their connection with these cells that degenerate, we have an important clue to the condition in any case of the cerebellar tracts. In lesion of the lumbar enlargement there is no degeneration; in lesion of the lower dorsal region few fibres are degenerated, and these are often difficult to find among the numerous normal fibres; besides a considerable number of the fibres of this part of the cerebellar tract run among the fibres of the lateral pyramidal tract. It is only in lesions of the cervical and upper dorsal regions that we have a compact band of degeneration. And yet not invariably even in these, for, in the second case reported, fracture of the cervical vertebræ with death in seventeen days, there was degeneration neither in the cerebellar tracts nor in Goll's columns, though there was distinct descending degeneration in the pyramidal tracts. In this case it is important to note the absence of the knee, abdomen, and cremaster reflexes.

(3) *On the Time of Appearance of Secondary Degeneration.*—Türck thought six months were required for the development of

secondary degeneration, but he was misled by regarding granule-cells as the only structures pathognomonic of secondary degeneration, whereas in point of fact the formation of granule-cells is consecutive to the degeneration, and is not a criterion of its presence or absence. Bouchard observed secondary degeneration fourteen days after compression of the cord, and sixteen days after an apoplectic attack. Barth found it five weeks after a cerebral lesion, and Müller thirteen days after a traumatic compression of the cord in the cervical region. Schiefferdecker, experimenting on dogs, noticed it on the fifteenth day, while Leyden, after inducing myelitis in dogs, observed degeneration of Goll's strands within a month. In the second case of compression of the cord (see *ante*) there was secondary degeneration seventeen days after the injury, and Kahler and Pick report another case in which secondary degeneration was observed eleven days after a lesion of the corpus striatum, optic thalamus, and external capsule. These cases cast doubt on Leyden's theory that the degeneration after traumatic or primary lesions of the cord develops more rapidly than after brain lesions. Some have thought that the ankle-clonus is dependent on secondary degeneration. But this view is untenable, for Westphal in a case of apoplexy with hemiplegia and contracture, observed both knee and ankle phenomena, which had previously been absent, one hour after the attack. Besides there are cases of secondary degeneration in which it is impossible to evoke the phenomenon of ankle-clonus.

W. J. Dodd, D. Sc.

(*To be continued.*)

Cortical Lesions of the Cerebral Hemispheres. (*Gazette des Hôpitaux*, January 24, 1880.)—Dr. H. de Boyer has presented a thesis to the Faculty of Paris, entitled 'Clinical Studies on the Cortical Lesions of the Cerebral Hemispheres;' and Dr. Gaston Decaisne, 'On Cortical Paralyses of the Superior Extremity—Brachial Monoplegia.' The following are the principal conclusions deduced from clinical observation, more particularly in reference to treatment.

First. *Of the principal cortical lesions; the determination of the latent zone and motor areas.* One of the most important of M. de Boyer's inferences is that which treats of the existence of latent tracts, non-motor or neutral, by the side of the motor areas. Cerebral lesions do not necessarily cause motor disturbances, it depends upon their situation. It is important to recognise the latent areas, because we thus can circumscribe these non-motor tracts, and by

exclusion demonstrate the existence and seat of a motor area on the cortex.

It appears already demonstrated that the motor centres occupy the circumference of the fissure of Rolando and the neighbourhood of the fissure of Sylvius. The localisation of aphasia consists of three lesions—of the cortex of the third left frontal convolution, of the island of Reil (at present under observation), and of the pediculo-frontal fibres of the third left frontal. The centre for language is in the posterior third of the third left frontal, and round the ascending ramus of the Sylvian fissure.

The lesions of partial epilepsy are never found in the base of the brain. Cases of monoplegia are alone sufficient to enable us to fix the exact seat of a centre. Associated monoplegias indicate the extension of a centre—whether this influence of one motor centre on another takes place by the superposition of the edges of two contiguous centres, or whether the white fibres derived from the two decussate in the interior of the hemisphere. We may observe the following monoplegias: aphasia, facial, brachial, or more rarely crural monoplegia, ptosis, rotation of the neck, or deviation of the eyes. These may be associated—the speech with the face, the face and the arm, the arm and the leg; but never with a single circumscribed lesion do we see monoplegia of the face and the leg, or the speech and the leg. The centre for the arm occupies the space between that for the face and that for the leg. We often find monoplegia of the arm associated with other monoplegias, most frequently with that of the leg, constituting incomplete hemiplegia, without facial paralysis. The centres thus range themselves from below upwards, along the fissure of Rolando, and govern their respective muscular masses in an inverse order; thus, the area for the face is lowest in the brain, while that for the leg is highest. The following centres may be fixed: Language, the posterior third of the third left frontal. Face, the inferior frontal and ascending parietal. Arm, the middle third of the frontal and ascending parietal. Leg, the superior third of the ascending parietal.

Clinically we may observe the following paralyses, corresponding to a single lesion: Loss of language; paralysis of the face; of language and face; of the arm; of the arm and the face; of the arm and loss of language; of the arm and the face and loss of language; of the leg; of the leg and the arm; of the leg, of the arm and the face; of the leg, of the arm and loss of language; of the leg, of the arm, of the face, and of loss of language.

Second. *Paralysis of Cortical Origin of the Upper Limb* (*Brachial Monoplegias*).—This study is based on clinical observations chiefly, and the following are M. Decaisne's principal conclusions :—

In general there is no apoplectic stroke, properly so called ; there is merely a giddiness, a trembling or loss of power, and the patient retains a more or less distinct idea of what is passing. Often there is a temporary embarrassment of the speech, and the leg first attracts his attention, the weakness of the arm not being noticed for a little time. The general symptoms soon dissipate, and the arm is left alone affected. The most striking symptom is the conservation of the sensibility. It is merely blunted, and that at the first onset. Should sensation not soon be recovered, it argues an extension of the cortical lesion. Sensation to heat and cold remains intact, and the temperature of the limb is unaffected. Most frequently the paralysis is incomplete, merely an enfeeblement. Usually, having been at the first onset general, it eventually settles in certain groups of muscles—e.g., the flexors or extensors, most commonly in the latter, or in certain segments of the limb, the forearm, wrist, or fingers. Inversely, the paralysis may progressively attack other groups of muscles than those first affected ; thus developing into a hemiplegia. The lower part of the face may be temporarily paralysed in this disease ; but should the symptoms extend to the upper part, it is no longer a case of brachial monoplegia. Should the lesion be permanent, motion may be re-established by the assistance of neighbouring cells. The prognosis depends on the extent of the lesion and its cause. If the lesion be limited, if it be a thrombosis, embolism, or hæmorrhage affecting a limited vascular supply—if the affection be purely local—a speedy recovery is the rule ; and if, on the other hand, the lesion involves the whole group capable of taking the place of the destroyed cells, it is irremediable. Should the first cause be such diseases as cancer, tubercle, &c., the prognosis will of necessity be bad, even with a very limited lesion ; but an extensive alteration may be quite remediable as in syphilis.

In these cases, then, the characteristic symptoms are the limited, incomplete, and transitory nature of the paralysis, the preservation of sensation, and the normal temperature of the affected part.

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// BRAIN.

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Original Articles.

ON TEMPORARY PARALYSIS AFTER EPILEPTIFORM AND EPILEPTIC SEIZURES; A CONTRIBUTION TO THE STUDY OF DISSOLUTION OF THE NERVOUS SYSTEM.[1]

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In the Mirror of the 'Lancet,' December 13, 1873, and October 26, 1878, are reported, with remarks, cases of temporary local paralysis after epileptiform convulsions; after those convulsions, that is to say, in which spasm begins in the terminal part of a limb or in the side of the face: these convulsions were first described by Bravais in 1824. As I there stated, the paralysis is supposed to depend on exhaustion of nerve-fibres consequent on the excessive discharge in the paroxysm. I do not now, however, speak so confidently as to the geographical position of the fibres exhausted; physiologically speaking, they are, I suppose, those connected with the discharging cells, and are presumably some fibres of the internal capsule. Since the remarks in the first report

[1] The term Dissolution has long been used by Herbert Spencer as the opposite of Evolution. I use it in that sense in this, as I have done in former papers. Great objections are made to this application of the term, but I submit that it is inexpedient to coin a new word for the opposite of Evolution of the Nervous System, when Dissolution has been used for at least fifteen years for the opposite of Evolution in general. The term is none of my choosing; it was used as I am now using it, long before I thought of the process it names.

appeared, I have found that the hypothesis advanced is in all essential respects the hypothesis of Todd and Alexander Robertson. It is briefly that the temporary post-epileptiform paralysis depends on temporary central exhaustion consequent on the excessive discharge during the paroxysm. During the paroxysm there is a severe nervous discharge (liberation of energy) beginning from highly unstable cells ("discharging lesions"[1]) of some portion of the cerebral cortex; the currents developed of course "flow" in the order of lines from those of least to those of most resistance; in other words, the movements of the external parts represented are developed in the order of their speciality of representation by the nervous arrangements (cells and fibres) of the portion of the cortex in question. Spasm in convulsion is nothing more than a contention, or sequence of contentions, of many of the movements represented by unstable cells of some part of the cerebral cortex; numerous movements being developed in a short time.

The convulsion is the external positive event answering to the internal positive event of excessive nervous discharge (great liberation of energy). The local paralysis remaining when the convulsion is over, when the discharge has ceased, is of the parts first and most convulsed, and is supposed to be the external negative condition answering to the internal negative condition of exhaustion of some fibres overworked from the excessive discharge of cells connected with those fibres. The nerve-fibres being only exhausted, not destroyed, as in some other kinds of paralysis, post-epileptiform paralysis is temporary, unless, of course, the fit recurs soon.

The condition following an excessive cerebral discharge is supposed to be analogous to exhaustion, by strong faradization, of a nerve going to the cut-off leg of a frog. A

[1] The very simple expression "discharging lesion" has, I find, led to difficulties. It has been taken to mean that some morbid product discharges, just as the expression "the kettle boils" might be taken to mean that the copper vessel and not the water boils. A "discharging lesion" consists in some alteration (excess of function) of some part of the patient; certain of his nerve-cells, representing some movements of some parts of his body, have by some pathological process become highly unstable; on their discharge, the movements they represent are suddenly developed in great numbers in a very short time. The contention of these movements is spasm.

closer analogy is the temporary paralysis of a frog after a tetaniform attack induced by a large dose of strychnia; the functional power of the motor nerves is temporarily destroyed. Since after an enormous dose of strychnia there is paralysis without prior convulsion, it is believed that strychnia may act directly on the motor nerves. However, Vulpian concludes:[1] "Les nerfs d'un membre privé de circulation chez une grenouille strychnisée perdent donc *une partie* de leur motricité sous l'influence des excitations prolongées, répétées et violentes, qu'ils subissent dans ces conditions." A still closer analogy is that, on strong faradization of a frog from mouth to anus there is a tetanic condition, and after it a *temporary* "résolution flasque et générale de toutes les parties du corps."[2]

The above is not the accepted explanation. Some physicians believe the temporary post-epileptiform paralysis to result from the cerebral congestion of asphyxia induced by arrested respiration in the paroxysm. In reply, it may be said that such congestion being universal in the brain cannot account for *local* paralysis. Again, the paralysis has been attributed to small extravasations of blood caused by congestion from arrested respiration. But the paralysis is of the parts first and most convulsed in the prior paroxysm, and it would be very remarkable if the extravasations in each succeeding fit always occurred in the same centre—in that representing the parts first and most convulsed. I do not adduce against this opinion the fact that the paralysis is temporary, for I admit that small clots may produce only temporary paralysis. There are other strong arguments against the accepted explanation. We find local paralysis after some epileptiform seizures in which there has been no interference with respiration whatever; paralysis so occurring cannot be caused by cerebral congestion, nor by extravasations in the nervous centres. On the other hand, local paralysis does not occur after severe seizures of epilepsy proper, in which there often is extreme asphyxia from spasm of respiratory muscles. To repeat; absolute, although temporary, local paralysis may be found where there has been no asphyxia in the prior paroxysm, and is not found after paroxysms of epilepsy proper when

[1] Arch. de Phys. 1870, vol. iii. p. 128. [2] Vulpian.

the asphyxia has been suddenly induced and extreme in degree.

It is asserted by some authorities that the local paralysis after epileptiform seizures is *not* always of the parts first and most convulsed. In my experience it always has been so. This being no answer, I can only ask that further observations may be made on the matter.

It is understood, of course, that we are speaking of paralysis following seizures which we may call chronic; we are not speaking of cases of meningeal or cerebral hæmorrhage; in some cases of cerebral hæmorrhage the clot which destroys some part of the brain, and which in this way produces hemiplegia, also causes convulsion. We speak of cases such as those in which a small tumour or other "foreign body" in the mid-cortical region of the brain leads to instability of cells near it[1]—leads to what I have called a "discharging lesion"; the cells rendered unstable occasionally "explode" or liberate much energy, or, in other words, discharge excessively, and all of them much more nearly simultaneously than the comparatively stable cells do in health. After their excessive discharge has ceased, as signified by cessation of spasm, there often *is* paralysis, and that paralysis is temporary.

It is supposed that the following is the sequence from a growth in the cortex to the temporary post-epileptiform

[1] I have suggested that the size and shape of cells, as well as their nearness to the tumour, or other source of irritation, will have to do with their becoming unstable; other things equal, the same quantity of matter in many small cells will present a vastly greater surface to the contact of nutrient material than the same quantity in one large cell. I have also suggested that small muscles, or, more properly, movements which require little energy for the displacements they have to effect (those of the face and of the hands in touch, for example), are represented by small cells. Such movements are rapidly changing during many of the operations they serve in—writing, for example—and require repetitions of short liberations of energy, and necessitate quick recuperation of the cells concerned. Movements of the upper arm are, in comparison, little changing, and require persistent steady liberation of energy. That small muscles are represented by small cells is not altogether a mere hypothesis. The masterly researches of Bevan Lewis seem to me to show that those parts of Hitzig and Ferrier's region which especially represent small muscles have most small cells. It is to be noted that parts like the hand will be represented in a great number of different movements, and therefore by numerous ganglion cells. It may be mentioned, too, that epileptic and epileptiform seizures usually begin in small muscles, and it often happens that for a time a patient's seizures are limited to small muscles.

paralysis. (1) A growth, often a syphilitic one, in the cortex cerebri; so far apparently there are no convulsive or paralytic symptoms. But the growth, in its character as a foreign body, (2) induces (presumably slowly) changes of instability in nerve cells near it. Still there may be no outward signs of it, or rather of these changes it induces. Next (3) some day the unstable cells discharge and produce a convulsive paroxysm. (4) After the paroxysm there is paralysis of the parts which were first and most convulsed. (5) The patient is apparently well again in a few hours or days, and keeps so until the next fit comes. (Of course the "foreign body" may produce the general symptoms of tumour, optic neuritis, headache, &c., which do not here specially concern us.)

It is recognised nowadays, thanks especially to Hitzig, Ferrier, and Charcot, that tumours in the mid-region of the brain, when very large and when much softening is induced, may be the cause of some permanent paralysis; here the paralysis is owing to permanent destruction. In this paper temporary paralysis after convulsion is alone considered, and so far only local temporary paralysis after epileptiform seizures.

It is not denied, of course, that convulsion may occur in a part which is already paralysed, partially or completely. For example, a patient may become hemiplegic without convulsion, and then on partial, or, indeed, on apparently complete, recovery, he may become subject to convulsions beginning in the hand, side of face, or foot. This I believe happens most often when the hemiplegia has resulted by embolism or thrombosis.

Cases of transient paralysis not preceded by convulsion, however often repeated, and however much like one another the recurring palsies may be, do not come here under our notice. Transient paralysis may be owing to a small clot, or to softening from blocking of a small vessel. Repetitions of similar local paralyses, each transitory, are hard to explain; but, as above said, we are not here concerned with them. Again, a patient subject to localised convulsion may have a batch of them, and in the intervals of the convulsions the parts occasionally severely convulsed may remain absolutely

paralysed. It is very singular to witness spasm beginning in and spreading over a limb which the patient can at no time move in the slightest degree.[1] In this paper, I repeat, temporary paralysis after convulsion is alone considered.

Let us note the sequence of phenomena in a very simple case, recorded in the 'Lancet,' October 26th, 1878, intercalating hypothetical explanations in brackets, and thus keeping them separate from the facts. A patient subject to epileptiform attacks [having permanently a local "discharging lesion"] has no obvious paralysis [since if the nervous arrangements, their cells being highly unstable, are useless for normal function, there is large Compensation by neighbouring parts]. One afternoon she has a fit [an excessive discharge, beginning from the part of the cortex the cells of which are unstable], the spasm affecting chiefly the right arm [brutal, that is excessive development and more nearly simultaneous development of the movements which the unstable cells discharging represent than would occur in healthy operations]; after the fit the arm is *absolutely* paralysed [exhaustion of central nerve-fibres has been effected by the discharge]; all paralysis is gone in five hours [there being only exhaustion, recuperation is soon complete].

Of course there are cases in which the post-epileptiform paralysis is trifling, and more transitory than in the case above instanced; the patient may say his hand is a "little numb," and we may find that he grasps firmly, but cannot pick up a pin or button his shirt. It is convenient to take for illustration a simple case; the reasoning is essentially the same. In the case instanced we have not merely to account for (1) local paralysis after a localised convulsion, but for two other things also; (2) for the absoluteness of the paralysis; (3) for the paralysis being very transient.

We have so far, except in passing illustration, spoken only

[1] Perhaps it may seem that such cases are counter to the hypothesis. But that the comparatively slight discharges during "volition" should fail to move a part, and that an excessive discharge should move it, is not extraordinary. Yet if the fibres be exhausted, how is it that by them the excessive discharge can effect convulsion of the part they pass towards? We must suppose that they are either not absolutely exhausted, or that they soon recuperate enough to be affected by the excessive discharge.

of epileptiform seizures. Logically, if Todd and Robertson's hypothesis is to be sustained, there ought to be paralysis after seizures of epilepsy proper. It would usually be said that there is none. So that whilst after the comparatively slight seizures called epileptiform there is often decided paralysis, there is, according to current opinions, none after the much severer paroxysms of epilepsy proper. At first glance this seems decisive against Todd and Robertson's hypothesis, as well as against the hypothesis of congestion and extravasation already combated. But local paralysis (of the face, or of a limb, or of one side of the body) is not the sole paralysis met with: there is such a thing as paralysis spread all over the body. The patient, after a severe paroxysm of epilepsy proper, has not, it is true, any paralysis of but one limb; he is not hemiplegic. He is, however, I submit, biplegic, if such a word may be coined. I mean that there is some, however slight, paralysis of both sides of the body; the paralysis is nearly evenly, I do not say absolutely equally, spread on both sides of his body. How does it happen that such paralysis is ignored? One reason I submit is, that being slight, evenly spread, and transient—there being no difference, or no obvious difference betwixt the two sides of the body—it is not easily recognisable as paralysis. Another reason is, that here, as in other cases, abnormal physical conditions are erroneously "explained" as being owing to abnormal psychical states. After a very severe fit of epilepsy proper, the patient does not move his limbs; all admit that. He seems a mere breathing, circulatory, &c., mechanism, lying otherwise inert on the floor. As he is then comatose, it would generally be said that he does not move *because he is unconscious.* A moment's consideration, however, will show that this is an explanation which verbally explains everything, and yet in reality explains nothing. It may pair-off with such a pseudo-explanation as that a patient does not move a limb because there is loss of volition, or with that which accounts for an aphasic's inability to speak, or for his speaking badly, by saying that it is "*because* he has lost the memory of words." It belongs to a whole family of psychologico-materialistic confusions; it is akin to that which has it that ideas or sensations (psychical

states) produce movements (physical states); to that also which declares that the mind affects the body. Nothing is said against the popular or clinical use of these expressions. No one objects in a clinical conference to the expressions that ideas or emotions affect, or, more inclusively, that the mind affects the body, when it is understood that what is meant is that centres, *during activity of which mental states arise*, can affect other centres, and thus the body. But much is to be said against the use, or rather abuse, of such expressions in what purport to be scientific explanations. It is, to say the very least, inexpedient in a scientific exposition to attempt to explain physical conditions by invoking crude popular psychological doctrines.

To return to the case. I submit that the post-epileptic comatose patient does not move, for the very simple reason that there is some negative *physical condition* of his nervous centres. It is an intelligible, if it be an erroneous, explanation to say that his highest nervous arrangements, and no doubt many lower ones too, are exhausted by the excessive discharge in the prior paroxysm. Surely the patient's not moving, if it be not owing to exhaustion of nervous arrangements, is at any rate owing to *some* negative *physical* condition; this negative physical condition is not loss of consciousness, but is only correlative with that negative *psychical* condition, if one may be allowed to speak of two negative conditions being correlative. I submit that it is not an intelligible explanation to say that the patient does not move because he is unconscious. Why should he not move if unconsciousness were all, his nervous system being sound? The fact is, he cannot be unconscious without having some negative physical condition of his nervous system answering to that negative psychical condition, and it is the central negative physical condition alone which we have to take count of in our explanations of his other physical condition of immobility.

The paralysis after a paroxysm of epilepsy proper, although widespread, is not necessarily, I suppose never is, total anywhere; it varies in different cases from slight to severe. In some cases of post-epileptic coma there is considerable movement on disturbing the patient. This is not in the least

counter to the hypothesis, for besides the possibility that but few of the patient's highest nervous arrangements are exhausted, many others, and also some of lower centres, are capable of functioning. There is no *à priori* reason why there should not be slight quasi-trifling paralysis spread all over the body—in other words, loss of some movements of every part of the body, with retention of many other movements of every part of the body—than that there should be slight quasi-trifling paralysis throughout an arm as there often is—in which case there is loss of some movements of the limb as a whole, with retention of many other movements of it. To speak of a part as being both paralysed and also as being movable by the patient, is not even unusual in ordinary clinical language. For to say that an arm is partially paralysed means that there is both some paralysis and also some movement remaining. We should not speak of paralysis of *muscles* from central lesions. Nervous centres do not represent muscles, but, or except as, movements. Hemiplegia appears as loss of power in certain muscles, but really is a loss of a number of movements of the parts paralysed. Similarly a convulsion appears as spasm of muscles, but is really a development of numerous movements of the parts, or rather an attempt at such development; spasm being a contention of many movements. From a small destructive lesion of a centre a few movements of muscles may be lost, and many other movements of the same muscles be retained; the hemiplegic man, who cannot easily button his waistcoat, and yet can lift a chair, has only lost some power in the muscles of the arm, in the sense that he has lost some of the most special or "delicate" movements of this limb. Similarly there may be loss of some of the most special or "delicate" movements all over the body, with retention of very many more general or "coarser" movements all over the body.

I do not shirk the logical consequences of the doctrine that the highest centres are, like all lower centres, sensori-motor—that from loss of function of more or fewer of them there is paralysis. Let me go into detail. I believe that in every case of insanity, however slight it may be, there is, so long as it lasts, defect[1] of consciousness. I believe, too, that there is neces-

[1] I dare say it may be denied that in some slight cases of insanity there is any

sarily correlative with this negative psychical condition, a negative physical condition of the highest nervous arrangements. Putting this otherwise, consciousness in health attends, but is not activity of these highest nervous arrangements; and when the function of them, or some of them, is lost by any pathological process, there is correspondingly loss or defect of consciousness. The highest centres are not consciousness, they are only the anatomical substrata of consciousness. Since the anatomical substrata of consciousness are, like all other centres, made up of sensori-motor arrangements, there should be widespread, however slight, paralysis in every case of insanity. I believe there is. Of course, in many cases, it would be difficult or impossible to demonstrate any. Some of those who might deny there to be any in a case of insanity might nevertheless admit it by saying that there was loss of expression, shambling gait, and slowness of movement. To take an extreme case, one of temporary acute mania after an epileptic paroxysm. The furious post-epileptic maniac is unconscious as well as maniacal. His symptomatic condition is double; there is a negative and there is also a positive element. I believe there is during his mania widespread paralysis consequent on temporary exhaustion of many of his highest nervous arrangements, which exhaustion is the negative physical condition answering to his negative psychical condition. I do not mean that his maniacal actions are the outcome of the exhaustion; that is an impossible explanation; there is supposed to be paralysis answering to that exhaustion. How then do we account for the co-existing over-movements of mania? The case is an example of Dissolution, using this term as the opposite of Evolution.

Evolution does not simply imply a progressing increase of complexity and speciality only, but also that the higher (more complex and special) nervous arrangements control or inhibit

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defect of consciousness, partly because it is erroneously inferred that what is clinically called insensibility is meant. Yet probably some of those who would deny defect of consciousness in a slight case of insanity would tacitly admit it by saying that the patient's judgment, power of attention, emotional control, or will was defective. A person has not got a consciousness in addition to will, memory, and emotion; these are only names applied to artificially distinguished aspects of consciousness; defective judgment, &c., is defect of consciousness.

the lower. There are two things: "the adding on" of the higher is at the same time "a keeping down" of the lower. Now as to the reverse process of Dissolution. It also is a double process. "The taking off" of the higher is a "letting go" of the lower. On removal of the influence of some of the highest nervous arrangements (on loss of their function in the case of post-epileptic mania by exhaustion), the next lower nervous arrangements, no longer controlled (Anstie, Dickson), spring into activity, and it is from *their* activity that the maniacal movements result. On the physical side there is loss of function of some of the highest nervous arrangements, and increased activity of the next lower. Correspondingly on the psychical side there is loss of consciousness and mania. In all other cases of insanity there is a negative and a positive condition, in some, as in post-epileptic mania, the positive condition is hyperpositive.[1] There is no inconsistency in supposing that there is paralysis in the sense of there being loss of some of the most special movements all over the body, and that there is also retention of very many others, and over-increased activity of them, any more than there is in supposing there to be loss of some movements of the arm in cases of chorea, where other movements of that limb are over-developed. I submit that what has

[1] I consider that there are many degrees of Dissolution consequent on exhaustion of different amounts effected by epileptic discharges of different degrees of severity. To make, perhaps with some arbitrariness, three degrees, there is (1) post-epileptic "ideation;" (2) post-epileptic action (mania for an example); and (3) post-epileptic coma. This is using ordinary nomenclature, which is misleading. There is a negative and a positive element in each degree. Along with post-epileptic ideation there is—(*a*) defect of consciousness; with the post-epileptic actions, there is (*b*) loss of consciousness, and in the third degree there is (*c*) coma. We compare these three negative conditions, saying they signify respectively Shallow, Deep, and Deepest Dissolution, the central condition being "shallow," "deep," and "deepest" exhaustion of nervous arrangements of the highest and perhaps also of lower centres. The positive elements comparable are (*a*) the "ideation," (*b*) the actions, and (*c*), not the coma, but the vital operations, circulatory, respiratory, &c., going on in the comatose state. These are owing to activity of nervous arrangements which are healthy except for being uncontrolled by their now exhausted higher nervous arrangements. Unless the duplex conditions in each be recognised, the three degrees cannot be shown to be analogous, nor can the cases be shown to come under the principle of Dissolution of the Nervous System. It is understood, too, that what we really compare and contrast are the physical conditions in the several degrees.

been called defect of voluntary co-ordination of the arm in chorea, is simply loss of so many movements of the limb, that it is so much paralysis, and what has been called morbid involuntary co-ordination, is simply over-development of some others. Besides we daily see co-existing the diametrically opposite conditions of paralysis and spasm, as in the rigidity of hemiplegia. So we do more rarely in the tremor of disseminated sclerosis, in which there is loss of some movements and retention of some others.

As aforesaid, the immobility of the post-epileptic comatose patient, which is here called paralysis, is commonly "explained" as being owing to a psychical loss. It would be denied that there is any paralysis. Yet after all, what is denied in technical terms is admitted in simple ones. On return of consciousness after the paroxysm, or of some consciousness, whilst it would be said that the patient is not paralysed, paralysis is really admitted in the statements that he is "prostrated" or "weak" after his fits. Even after slight epileptic attacks (le petit mal) there is veritable paralysis; the patient is weak ("unfit for anything," "thoroughly done up," are the kind of expressions patients use). Let me say explicitly that if paralysis is to mean only local paralysis, the patient after a slight or severe epileptic paroxysm is *not* paralysed. I admit that most willingly, indeed I assert it. But it is not fair to make such a limit to the meaning of the term. I submit that the patient is paralysed. His "weakness" is veritable paralysis, even if it be owing especially to exhaustion of nervous arrangements for vital organs (cardiac, respiratory, &c.).

I venture then to submit (1) that the psychological "explanation" is really no explanation of the comatose patient's immobility; the explanation of a materialistic condition should be in materialistic terms. Our concern, as physicians, with negative or positive psychical conditions is simply to get to know what is going wrong in the *nervous system.* The absolute distinction betwixt mind and body is insisted on, not as some seem to suppose, because a psychological view is taken of any nervous diseases, but for the diametrically opposite reason. *The distinction is made in order that we may be thoroughly materialistic in our dealings with disease,* and that

we may methodically consider the nervous system from top to bottom as a mere sensori-motor mechanism.[1] Next I submit (2) that there is paralysis after epileptic seizures and that (3) this paralysis is universally spread, and (4) is owing to exhaustion of some nervous arrangements of the highest centres and perhaps of lower ones too.

That loss of function of nervous arrangements of the highest centres (which centres, although neither they nor their activities are consciousness, are yet the anatomical substrata of consciousness), should entail paralysis in the sense of universal weakness, is in accord with the doctrine of Evolution of the nervous system. And that it should not entail local paralysis is equally in accord with that doctrine. I have long held that the very highest centres are, like all other centres, sensori-motor. In other words, parts of the brain other than Hitzig and Ferrier's centres (lower cerebral centres) are, I think, made up of sensori-motor arrangements. I do not say this in consequence of their experiments; I urged it before their experiments were begun, and urge it still. For various reasons I think that the nervous arrangements of the highest centres are the most special and complex of all sensori-motor arrangements. Each unit of the highest centres represents, I believe, the organism as a whole (complexity) and each unit represents

[1] Scientific materialism is quite a different thing from crude popular materialism. Scientific materialism distinguishes betwixt mind and nervous system in order to study each thoroughly. Popular materialism does not separate the two, and mixes up in its "explanations" psychical factors and physical factors. Scientific materialism is only materialistic as to what is material, the nervous system. Popular crude materialism, making no distinctions, confuses two utterly different things, psychical states and physical states. It is said that in an early stage of paralysis agitans the patient can keep his hand still by an act of volition. No one objects to this phrase used clinically if the physical process corresponding to what is psychically volition be meant; if it be meant, for example, that a cerebral discharge can inhibit the movements, or inhibit spinal or other centres for them. But if it be really meant that The Will can arrest a physical process, the statement is most unwarrantable. It amounts to saying that the will influences matter. Let any one try to conceive how the will can stop a movement; it is on the other hand conceivable, if not believable, that a nervous discharge *during* "volition" may stop a movement, for both are material things. Clifford writes ". . . if anybody says that the will influences matter, the statement is not untrue, but it is nonsense. The will is not a material thing, it is not a mode of material motion. Such an assertion belongs to the crude materialism of the savage."

the whole organism differently (speciality). There is, to speak figuratively, "an experiment made by disease," which seems to me to demonstrate that the highest nervous arrangements represent the whole organism. In a severe paroxysm of epilepsy proper, one setting in with loss of consciousness (when of necessity, therefore, the corresponding physical process of discharge begins in some part of the highest centres) the whole organism is involved; there is universal convulsion. The convulsion in epilepsy proper is, I take it, a brutal development of movements represented in the highest centres primarily discharged, and doubtless too in lower centres secondarily discharged. The discharge "by disease" beginning in some part of the highest centres of either side of the brain shows that they represent movements of all parts of both sides of the organism; they show this as certainly as artificially induced discharge (by galvanism or faradism) of any centre in Hitzig and Ferrier's region shows it to especially represent movements of some external part. This conclusion from an "experiment by disease" is strictly in accord with the doctrine of Evolution. As consciousness, or most vivid consciousness, represents the whole subject (subject-object) so the anatomical substrata of consciousness, or in other words the highest nervous centres, represent the whole organism. There is still another way of looking at this aspect of the question.

In many cases of epileptiform seizures beginning by discharge of some part of the mid-region of one side of the brain, and developing first local one-sided spasm, the convulsion *becomes* universal. The difference betwixt such a convulsion from discharge of part of lower cerebral centres and one of epilepsy proper from discharge of part of the highest centre, is only a difference in Compound Degree; it is such a difference as great difference in Evolution of the two centres would lead us to expect. The doctrine of evolution implies that the highest centres of one side are evolved out of the lower cerebral centres of the same side; that they re-represent all the parts which the lower centres have represented but in more numerous, more complex, and more special combinations.[1]

[1] That both sides of the body are represented in each half of the brain is quite certain as "descending" wasting in certain cases of one-sided cerebral disease

The two convulsions, (1) epileptic from discharge of part of the highest centres and (2) epileptiform, from discharge of part of lower cerebral centres, are in accord with these statements. The severe convulsion of epilepsy proper is more quickly universal, the parts on the two sides of the body are convulsed more nearly simultaneously, and the different movements of every part are developed more nearly contemporaneously, than in the severe epileptiform seizure. That it is a difference of degree is shown, in part at least, by the fact that in most cases of epilepsy proper the convulsion affects one side a little earlier and a little more than the other.

Frequently, however, the epileptiform convulsion is limited; the spasm is local. Just as after localised convulsion (epileptiform seizure) there is local paralysis, so after universal or widespread convulsion, there is, I submit, universal or widespread paralysis. The explanation given of post-epileptic immobility is, it seems to me, at any rate as good as the psychological explanation; if, indeed, the latter deserves the name of explanation.

To return now to cases of patients locally paralysed after localised epileptiform seizures. It may be alleged against the hypothesis put forward, that there is no proportion betwixt the severity of the seizure (the severity of the discharge), and the subsequent paralysis (amount of exhaustion). But once more it is pointed out, that decided local paralysis is usually what is solely called paralysis. Widespread slight temporary paralysis is not easily recognised as paralysis. If a man has total weakness of one arm, or even slight weakness of it after an epileptiform convulsion affecting that limb, that weakness is called paralysis. But if there be universal transitory slight weakness after a seizure, it is not called, but really is, paralysis.

On the hypothesis, there ought logically to be an exact relation of proportionality betwixt the discharge and the subsequent central exhaustion; betwixt the convulsion and the subsequent paralysis. I submit that there is. The relation is,

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proves. And since the fibres wasted in the spinal cord are some of those of the opposite lateral column and some of those of the anterior column of the same side as the cerebral lesion, there is warrant for the inference that the two sides of the body are differently represented in each half of the brain.

however, not easy to see at first glance. I ask the reader to remember that I expressly admit, that after the severest epileptiform seizures, there is less paralysis than after the slight ones, *if by paralysis local paralysis be meant*, and that I expressly deny that there is less if paralysis be used, as I submit it ought to be, for widespread or universal weakness as well as for local weakness after a fit.

We must, in the remainder of this paper, discard the vague word "severe" as applied to nervous discharges in disease, and speak of quantities of energy liberable and rates of liberation. Different quantities of grey matter will be unstable in different cases, and there will be differences of degree of instability of cells in different cases. Thus, in different cases there will be different quantities of energy liberable and liberated in the paroxysms. It is quite a certain thing that these are all degrees of spasm in unilaterally beginning convulsions from slight paroxysmal twitching of a few fingers, or "starting" of the big toe, to the severest universally-becoming convulsion; hence there are different quantities of energy liberable. But we must consider not only the quantity of energy liberated by discharging cells, but the rapidity of its liberation. Of course the conditions are complex, different quantities of energy will in different cases be liberated in different times. We estimate the two things respectively, but of course very roughly indeed, by the amount of spasm, and by the suddenness of onset and rapidity of spreading of spasm.

I think as a matter of observation that, other things equal, the more deliberately spasm sets in, and the more slowly it spreads, the more local it is, and also the longer it continues; and further, that the paralysis after such spasm, is correspondingly more local, is greater in degree, and more persistent. If so, we may, on Todd and Robertson's hypothesis, say of the central process, that the more deliberately the discharge begins, and the slower it is, the less widespread are the nerve-currents developed, and the longer they continue; and that consequently the post-epileptiform exhaustion of centres is more local, more complete, and more persisting. On the other hand, the more suddenly spasm sets in, and the faster it

begins to spread, the greater, I think, is the range it attains; and further, that the paralysis after it is more widespread, less in degree and more transient. If so, we may, on Todd and Robertson's hypothesis, say, that the more suddenly the discharge begins, and the more rapid it is, the more widespread are the currents developed, and the less time do they continue; and that the post-epileptiform exhaustion of centres is more widespread, less extreme, and more transient. The paralysis, in the latter cases, is *apparently* less, because it is not so localised, because it is slighter in degree, and because it soon passes off—in a word, it is not easily recognisable as paralysis, and may yet be admitted to be so, being called weakness.

Suppose two patients, in each of whom the same cortical area is affected, and so affected in one that a certain quantity of energy is occasionally liberated slowly, say in ten minutes, and so affected in the other, that an equal quantity is occasionally liberated rapidly, say in two minutes. Let us suppose that the part of the cortex represents especially movements of the arm. I suggest that the currents developed in the first case might "flow" almost exclusively to the arm, to the parts most specially represented in the part of the cortex discharged, and that in the second case they would "flow" less exclusively to the arm, but also to parts more generally represented by it, and finally to lower and collateral centres.

The rapid and sudden spreading of spasm implies a sudden rapid liberation of energy, and thus a shorter liberation of what is liberable; the currents developed will have greater force than those from a slow liberation of an equal quantity of energy. It is not asserted that nerve impulses travel faster in cases of rapid liberation than in cases of slow liberation; but that there is in the former a greater "quantity of motion" in a short time against an equal quantity in a long time in the latter. The more rapid short liberation (since currents of greater force, although over a shorter time, are developed) will overcome greater resistances than currents of less force over a larger time from slow, lengthy liberation. As will be seen, I do not use the term force as synonymous either with momentum or energy; it is, however, difficult for me to use

the term force without misgivings, since, among other reasons, it is a term used differently by different people. Supposing every molecule of a nerve to be of the same mass, and to always travel or vibrate with the same velocity, the momentum (quantity of motion) of each impulse is equal. Suppose the quantity of energy liberated to be equal in two cases, the more rapid liberation will lead to a current in which a greater number of such momenta occur in a short time, against an equal number in a long time during the slower liberation.

The movements of external parts which any centre *most* specially represents are, physiologically speaking, those united to it by lines of *least* resistance. We must bear in mind that any centre represents external parts by lines of different degrees of resistance, and no doubt often represents parts indirectly by intermediation of other centres, lower or collateral. The more force resulting from liberation of energy upon nerve fibres, the greater are the resistances which can be overcome. When greater resistances are overcome of course the wider range has the spasm, and thus the more widespread will be the subsequent paralysis.

But why is there not, after rapid discharge, as much local paralysis in the parts which were first convulsed as after the slower discharges, as well as paralysis in those parts which are next convulsed? Let me put this again. Supposing there is such a discharging lesion that there occurs a slow discharge upon an arm leaving it completely paralysed. Let us imagine, simply in order to state the question more clearly, the cells of the discharging lesion to become so much more unstable, or many more to become unstable, that in the next paroxysm the discharge is much more rapid, and that the whole body is convulsed. Why in the latter case is not the arm as much paralysed as in the former case, although there is also some slighter paralysis of the rest of the body? As a matter of fact, I submit that the paralysis is really less in degree in the part first convulsed when the spreading of spasm is rapid, and goes beyond the part, than when it is slow and is confined to that part. There is less spasm of the part first convulsed, that is, of the parts most specially represented by the cells discharging. When by more rapid

discharges the next more resisting lines (of lower or collateral centres) *are* overcome they are *then* paths of less resistance than before. The energy when more rapidly liberated by the discharging cells may be said to escape in more ways; the currents developed are less concentrated on the external part which the discharging cells most specially represent; the currents developed overcome lines of next and next greater resistance; hence parts most specially represented in the centre are less convulsed than in slow discharge, and those less specially represented more than in slow discharges.

On the other hand, in slow discharges the currents developed will keep more to lines of least of all resistance, will "flow" to parts most specially represented; being unable to overcome lines of greater resistance, they will *continue* to flow only on the lines which can be most easily overcome; thus the spasm is local and lengthy.

If the above reasoning be valid we should expect after sudden rapid short discharges, no decided local persisting paralysis, but slight widespread temporary paralysis, often so slight, so widespread, and so temporary that it is ignored as paralysis and called prostration. If, to speak roughly, as much paralysis as makes one arm useless for four hours could be spread out thin all over the body for perhaps a quarter of an hour, it would not be recognised as paralysis at all, but as weakness, &c.

No doubt, after the more rapid discharges there is really more paralysis. The "discharging lesion" is like a fulminate; by the rapidity of the discharge it overcomes the resistance of the normally unstable (comparatively stable) cells of physiologically connected centres, and the more rapidly it discharges, the more of healthy and collateral centres is it likely to overcome; it will produce effects in more ways more nearly at once. The currents developed by a rapid local discharge having overcome healthy collateral or lower centres, the energy of these centres will be added on to that of the primal liberation; there will be a compound effect from a more rapid discharge.

PISCIDIA ERYTHRINA.

BY ISAAC OTT, M.D., EASTON, PENNSYLVANIA.

THIS drug has recently come into notice as a substitute for opium in cases where pain is present, as in neuralgia. The bark of the root is used in medicine, from which a fluid extract is made. This extract has an odour greatly resembling laudanum, a reddish colour and a peculiar warm taste. The tree grows in the arid districts of Jamaica, and may attain a height of twenty to thirty feet. The bark of the tree is smooth and of a bright colour. The natives of Jamaica call the tree the "fish-catching coral tree."[1] The method of catching fish is as follows:—the leaves, twigs and bark of the roots are macerated with the residue from the distillation of rum or lime-water. Then they are put in baskets, which are dragged up and down the ponds till the narcotic principle is extracted, when the fish are stupefied, and, upon rising to the surface, are caught and eaten with impunity. The small fish only are killed by the drug. In determining the physiological action of this medicine, I used frogs and rabbits. When a rabbit received twelve drops of the fluid extract of Piscidia subcutaneously, respiration became frequent, incoordination ensued, and the pupils dilated. When twenty-four drops were given subcutaneously, sensibility was blunted. When another dose of twenty-four drops was given subcutaneously, the strongest pinching did not move him; lay sleeping; saliva flowed; death ensued by paralysis of the respiratory apparatus—heart beating at the time of death one hundred and sixty-eight per minute. To determine the action of the drug on frogs and the circulation in rabbits, I added to

[1] 'New Remedies,' 1880.

an ounce of the fluid extract an equal quantity of warm water, and evaporated the whole quantity down to about five drachms. The temperature of evaporation did not exceed 175°. The object was to get rid of the alcohol, which would confuse the results. I shall allude to the product obtained by evaporation as an "infusion" of the drug. When a frog receives twenty drops of an infusion of Piscidia, he soon assumes a squatting posture, can be handled without any effort to escape, pupils are dilated, seems asleep, sensibility blunted. When twelve drops more of the infusion are given, he is unable to hop away; when pinched, tetanus supervenes; does not make any reflex movement upon pinching; death ensues. When the sciatic nerve is exposed, it is irritable to a very weak current. To study the effect on the sensory nerves I used the ligature. For reflex action, the method of Türck was employed. Upon the circulation, the changes were studied by means of Ludwig's kymographion. The results obtained by these methods are found in the table on page 454, where they are compared with those of morphia and chloral.

Compared with atropia, piscidia, unlike it, does not paralyse the motor nerves, it does not paralyse the chorda tympani; it does not arrest the sudoral secretion; it does not paralyse the pneumogastric; it does not elevate greatly the arterial tension, but, like it, dilates the pupil. In its action on a man in health, it reduces the pulse, causes salivation and sweating, disturbance of vision, itching of skin, sleep. It seems to be more powerful to relieve pain than to narcotise. In a case of aneurism of the abdominal aorta under my care this drug acted admirably in relieving the neuralgic pains without constipating or disturbing the appetite, two things which opium did. It sometimes, when first given, causes a slight nausea. To obviate this, it should be given on a full stomach, and Dr. F. L. Putt[1] recommends its combination with a small amount of glycerine, camphor-water, and a few drops of essence of wintergreen in syrup. The nausea that ensues after opium has been taken is seldom seen after the action of Piscidia. Neither does Piscidia leave a

[1] 'Therapeutic Gazette,' Sept. 1880.

general nervous disturbance, like opium. As a sedative, for the cough of different pulmonary complaints it has been found quite useful. That this drug acts somewhat peculiarly, due

Piscidia Erythrina.	Morphia.	Chloral.
It is narcotic.	It is narcotic.	It is narcotic.
It does not affect the irritability of the motor nerves.	It increases and then decreases the irritability of the motor nerves.	It does not affect the motor nerves.
It does not act on the peripheral ends of the sensory nerves.	It excites sensory nerves.	It does not affect sensory nerves.
It reduces reflex action on the centres of Setschenow,	It increases and then reduces reflex action.	It increases and then decreases it.
It produces tetanus by a stimulant action on the spinal cord.	It causes tetanus by a spinal stimulant action.	It convulses.
It dilates the pupil.	It contracts the pupil.	It contracts the pupil.
It is a salivator.	It dries up salivary secretion.	
It excites sudoral secretion.	It causes slight sweating.	
It reduces the frequency of the pulse.	Small doses slow the pulse and then accelerate it; large doses slow the pulse by an action on the heart.	It reduces the frequency of the pulse by an action on the heart itself.
It increases arterial tension by stimulation of the monarchical vaso-motor centre; this rise of pressure is soon succeeded by a fall, due to an action on the heart and the vaso-motor centre.	It increases and then decreases the arterial tension, due to stimulation and paralysis of the main vaso-motor centre.	It reduces arterial tension by paralysis of the main vaso-motor centre.

probably to the idiosyncrasy of the patient, is shown in a case reported to me.[1] A man with paraplegia, supposed to be syphilitic, was given at 8.30 P.M. a drachm of the liquid-extract

[1] Letter of Dr. C. E. Bean, Jefferson Medical College Hospital.

of Piscidia, which produced profuse sweating, with increased action of the heart, the pulse beating stronger and fuller, followed by a rapid and weak pulse. At 10.30 P.M. the dose was repeated, the same effect ensuing. At midnight a third drachm was given, and in about fifteen minutes the skin grew quite cold, respiration laboured, and pulse very feeble. He lay in a stupor two hours, after which he slept an hour. Repetition of doses of a drachm or two drachms on following nights caused no narcotic effect, but perspiration, nausea, with loss of appetite in the morning. From my knowledge of this drug in its physiological aspect,[1] and in the practice of my profession, I regard it as well worthy of a trial by the profession. The consensus of several good observers is needed to establish the proper therapeutic rank of this agent.

[1] 'Detroit Lancet,' June 1880.

CEREBRAL AMBLYOPIA AND HEMIOPIA.[1]

BY DAVID FERRIER, M.D., F.R.S.

Some important differences of opinion still exist respecting the relations of the eyes to the cerebral hemispheres, and the facts of clinical medicine and pathology appear in some respects neither to agree altogether with any of the views founded on physiological experiment, nor to be quite in accordance with each other. My object in this communication is to remove some of these differences, and to endeavour to explain some of the apparent discrepancies among clinical facts. As regards the relations of the eyes to the cerebral hemispheres, and the localisation of a visual centre, the question may be briefly stated to stand at present thus :—

In monkeys and in other animals, I, followed by Munk, Luciani and Tamburini and others, have found experimentally that destructive lesions of certain parts of the cortex cerebri cause affections of vision, and of vision only, other forms of sensibility and motor powers remaining intact. In monkeys I localised[2] the visual centre in the angular gyrus; and I have stated that unilateral destruction of the angular gyrus causes total loss of vision in the opposite eye, of a temporary character, however; while bilateral destruction entails total loss of vision in both eyes—an effect which I inferred would be of a permanent character. I also stated that the greater portion of both occipital lobes might be removed without any discoverable impairment of vision.

Munk[3] localises the visual centres in the occipital lobes.

[1] The substance of this Paper was read at the Meeting of the Brit. Med. Ass. at Cambridge, Aug. 1880.

[2] 'Functions of the Brain,' § 65.

[3] 'Verhandl. der Physiolog. Gesellsch. zu Berlin,' April 12, 1878.

He states that a destructive lesion of one occipital lobe causes hemiopia of both eyes—the retinæ being paralysed on the side of lesion, and therefore entailing blindness towards the side opposite the lesion. Bilateral lesions cause total blindness.

Luciani and Tamburini[1] agree with me, in opposition to Munk, in placing the visual centre in the angular gyrus; but they include also the occipital lobe, partly or wholly. They agree with Munk in stating that a unilateral lesion of the visual centre causes bilateral hemiopia, and not crossed amaurosis such as I have described. This is a very brief statement of the different views, but essentially it is all that needs be mentioned in the present relation. Goltz's facts,[2] apart from his views as to the localisation of function, also demonstrate affections of vision from cortical lesions.

Now if we turn from physiological to clinical and pathological observation, we not only have no clear evidence of impairment of vision from purely cortical lesion, but we have actually many cases on record, in which post-mortem examination has revealed destructive lesions in the angular gyrus, and in one or both occipital lobes, without any affection of vision having been observed during life.[3]

The clinical history and morbid anatomy of cortical lesions, therefore, if it does not oppose, at least does not give direct support to the localisation of a visual centre in the regions indicated. The evidence in favour of a cortical visual centre in man is mainly of a constructive character; that is to say, there are many clinical phenomena which are best explained by the hypothesis of such a centre, but they can scarcely be taken as at all clearly indicating its position.

Among the facts of this nature may be mentioned, the irritative symptoms, ocular spectra, coloured vision, &c., which occur in connection with "discharging lesions," situated towards the posterior aspect of the hemisphere; as in cases reported by Hughlings-Jackson and Gowers.[4]

[1] 'Sui Centri Psico-Sensori Corticali,' 1879.
[2] 'Pflüger's Archiv,' xx. 1.
[3] See the author's 'Localisation of Cerebral Disease,' p. 110 *et seq.*
[4] 'Lancet,' March 1879.

Secondly: the phenomena of "word-blindness" occurring in connection with lesion in the region of the angular gyrus, of the left hemisphere especially; as in a case related by Broadbent.[1]

Thirdly; the temporary abolition of vision in one eye in connection with meningo-encephalitis of the opposite hemisphere, as in a case related by Abercrombie.[2] With this may be taken also the unilateral affections of vision described by Fürstner in general paralytics.[3]

The facts relating to injury and disease of the brain in the occipital region are discussed subsequently.

Other considerations might be advanced in support of the hypothesis of a definite visual centre in the cortex; but I do not propose to enter farther into this question, which I have already discussed elsewhere.[4]

But if the relation between visual defects and lesions purely cortical is not in all respects sufficiently well demonstrated, it is otherwise as regards the effects of lesions in the internal capsule, and in the medullary fibres in the neighbourhood of the optic thalamus, corpora geniculata, and occipital region.

In cerebral hemianæsthesia of organic origin, depending, as was first indicated by Türck, on lesion of the posterior segment of the internal capsule external to the optic thalamus, and since abundantly confirmed by numerous other reported cases, vision, as well as other forms of sensibility, is affected in a very distinct and special manner.

In the classical form of this affection vision is greatly impaired, if not temporarily entirely abolished, on the side opposite the lesion; the field of vision is greatly contracted; and there is achromatopsy, or impaired colour-perception.

Generally also, as was first pointed out by Landolt, there is some contraction of the field of vision on the same side as the lesion; but the visual impairment, i.e. the diminished acuity of vision and dyschromatopsy is essentially a crossed one. I might quote many accurately investigated instances of this

[1] 'Med. Chir. Trans.' 1872.
[2] 'Dis. Brain and Sp. Cord,' p. 121.
[3] 'Archiv für Psychiatrie,' 1877 and 1879.
[4] Op. cit.

affection now on record, but it will suffice if I refer to the perimeters of two well-marked cases recently under the care of Dr. Hughlings-Jackson and myself, and notes taken by Dr. Beevor.

Case I.—T. H., aged 67, a house-painter. Never suffered from lead-colic or wrist-drop. No history of syphilis. Has had gout in the left foot three or four times.

The present illness came on suddenly seven months before admission into the National Hospital for Paralysed and Epileptic on March 30, 1880. He was engaged melting some lead, when he suddenly lost consciousness and fell down, without having had any premonitory symptoms. He regained consciousness in a few minutes, and found that he was paralysed on the right side of the body. His speech was indistinct, but he could understand what was said to him, and make himself understood in words. He says he lost both motor power and sensibility on the right side.

He first began to be able to move his fingers about a fortnight after the onset. In another fortnight he could move the elbow, and a little later he could move his arm at the shoulder.

He could not move the leg at all for two months. He first was able to flex the thigh, then the foot, then the leg, and lastly the toes.

Present Condition.—Patient is a strong, healthy-looking man. There is no cardiac disease, and the urine is free from albumen. Arcus senilis is well marked, and the arteries are somewhat rigid and tortuous.

Motility.—The right angle of the mouth acts a little less vigorously than the left, but the difference is not marked.

All the movements of the right hand and arm are possible, though feebler than on the left. The grasp of the right hand$=$ 2 kilos., that of the left$=$25 kil. There is, however, some stiffness of the phalangeal joints of the right hand, which impedes firm closure of the fist. There is a good deal of fibrillar tremor of the muscles of the right arm, especially the extensors, and there is well-marked oscillation—of the disseminated sclerosis type—when he endeavours to place his forefinger on a given mark.

The movements of the right leg are all capable of being carried out, though with much less vigour than the left.

Patellar tendon-reflex is exaggerated on the right side. There is no ankle clonus.

Sensibility.—Sensibility, both general and special, is impaired on the whole of the right side.

Face.—There is complete abolition of sense of impressions of

contact on the right side of the head and face. Painful impressions are felt only on the face and head in front of a line drawn vertically through the auditory meatus. Pressure over the exit of the branches of the fifth, causes severe pain. The ear may be pinched severely without causing any sensation.

Arm.—He cannot distinguish two points on the right hand. Tactile and painful impressions on the forearm are referred to the palm of the hand. Painful impressions, as such, are recognised and correctly localised on the fingers.

Leg.—Tactile impressions are not recognised anywhere on the leg or foot, but painful impressions are felt and fairly localised.

Muscular Sense.—Tested with balls alike in appearance, but of different weights, he can distinguish between heavier and lighter balls; but underestimates the absolute weight with the right hand, on the average making the same weight with the left hand double what he makes it with the right.

Smell is completely abolished in the right nostril. There is also almost complete anæsthesia in the mucous membrane of the right nostril.

Taste is abolished on the right half of the tongue. There is also tactile anæsthesia of this side, and of the mucous membrane of the right half of the mouth and palate.

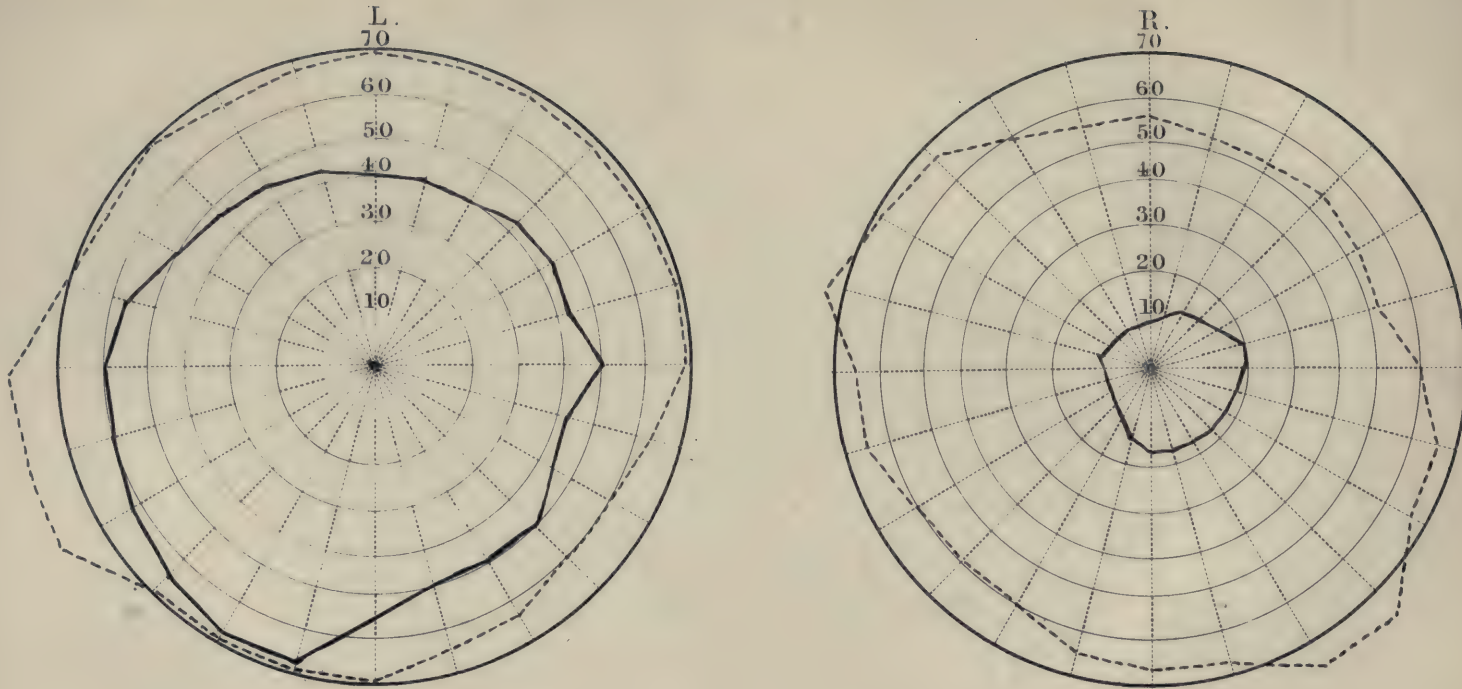
Hearing is defective on the right side. With Gellé's aural tube, he hears a watch at 15 inches on the left side, and at 8½ inches on the right.

Vision.—Acuity of vision normal in left eye.

Vision is dim and indistinct in the right eye. Tested with Snellen's test types, he recognises E. No. CC. at 3 feet; C. (No. C.) at 2 feet, and LXX. at 1 foot. He requires to move his head in order to take in the whole of E., as he cannot see it all at once. Holmgren's coloured skeins he recognises and names fairly correctly.

The perimeter (see Plate I.) taken on April 24 shows that the field of vision is contracted on the right side to an almost central area—slightly towards the right—measuring from 10–15 degrees. The field of vision in the left eye measures 50 degrees from the centre towards the right, and 60 degrees to the extreme point of the left. (The want of exact symmetry depends on the prominence of the nose.)

On June 5, along with slight improvement in respect to the



C. Berjeau.

Mintern Bro's, imp

other defects, the perimeter showed that the field of vision in the right eye had extended to 60 degrees from the centre all round, and slightly farther upwards and to the left and downwards and to the right. In the left eye the area had extended to 70 degrees all round—the limit of the normal chart.

The ophthalmoscopic appearances of both eyes were in every respect normal.

Case II.—Hannah O., aged 40, married. Has had eight children, five of whom are now alive. Four years ago, when in a state of anxiety about her daughter who was very ill, she went to bed quite well one night, but had an attack in the night in which she became insensible.

Was confined to bed for a week, and was more or less ill for six weeks, but not paralysed as to any of her limbs.

At the end of March, 1880, after a heavy day's work in the kitchen, she felt giddy in going upstairs. Shortly after she became insensible, and continued so for four days. When she came to herself, found she had lost feeling on the left side, and could not move the left arm or leg. This continued for a fortnight, at the end of which she began to move the fingers a little. The foot recovered later, but she was not able to walk without support for five or six weeks. During this time, she accidentally discovered one day, on closing her right eye, that she could not see with the left eye. Since her last attack she has continued feeble on the left side, particularly the leg, the condition of which she alone complained of when she came to the National Hospital for Paralysed and Epileptic on June 4, 1880.

Present State.—Patient is a fresh-looking woman. There is no cardiac disease, and the urine is free from albumen.

Motility.—The left side of the face acts very feebly as compared with the right.

The movements of the left arm and hand are feeble. Grasp of left hand = 5 kilos., that of the right = 25 kilos.

The left leg is very feeble, and the movements of the foot paralysed. In walking, she rubs the toe of her left boot on the ground.

Sensibility.—Muscular sense. Has a very imperfect appreciation of the position of her left arm when her eyes are shut. When she was asked to touch her nose with her left hand, she made an effort, and thought she had done it, though the arm, being firmly held was not moved. When tested with differently weighted balls, she

estimated 20 oz. in the left hand, as of exactly the same weight as 3 oz. in the right.

Cutaneous Sensibility.—She cannot feel a slight touch (such as is distinctly felt on right side) anywhere on left side. With stronger pressure, she feels and can localise fairly well, but she does not complain of pain on pinching or pricking deeply. Powerful stimuli are felt as mere contact. She cannot appreciate two points at the greatest distance possible, on the face, or hand, or foot.

Smell.—Smell is entirely abolished in the left nostril. The mucous membrane is also anæsthetic.

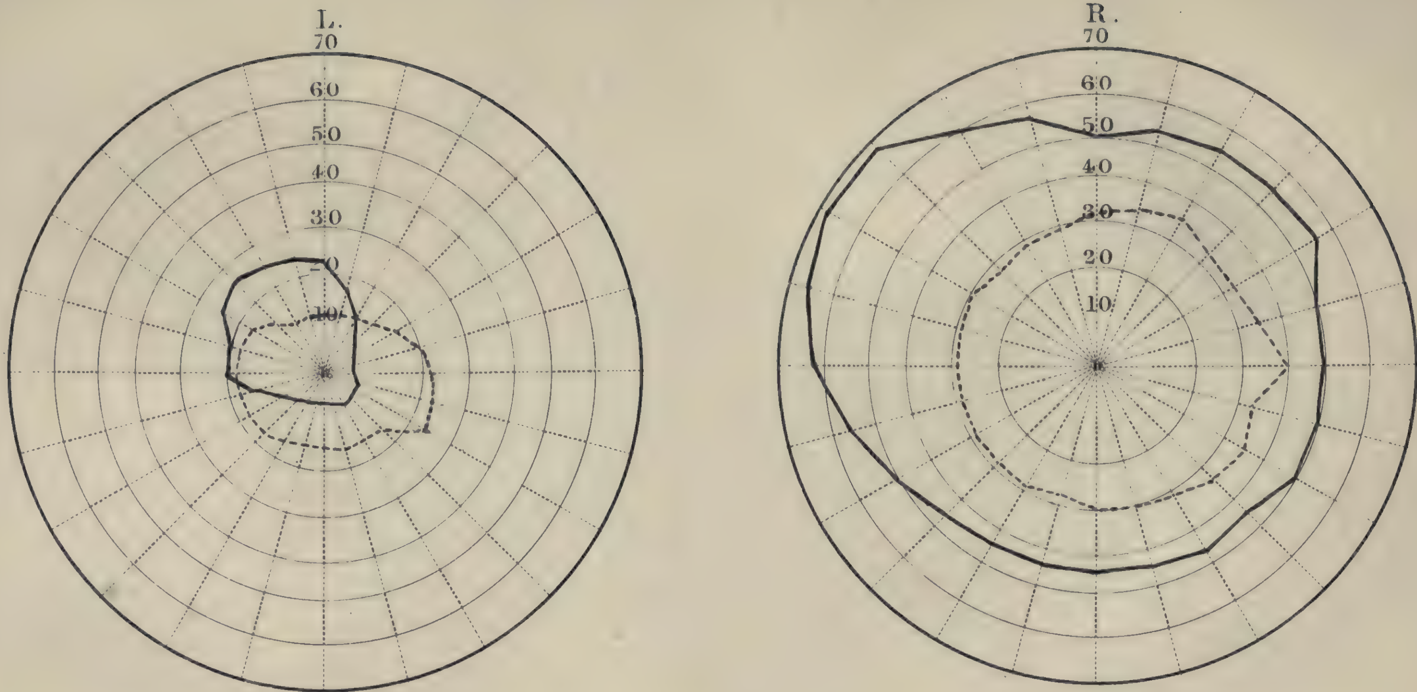
Taste.—Taste is completely abolished on the left half of the tongue. There is also abolition of tactile sensibility on the left half of the tongue, and on the left side of mouth and palate.

Hearing.—Is entirely abolished in the left ear. She cannot hear a watch or tuning-fork with this ear, either closely applied to the ear or conveyed through the skull. With the right ear she hears (through Gellé's tube), the tick of a watch at 60 cm.

Vision.—There is no ptosis. The refractive media and ocular tension normal, and the fundus and optic discs normal in both eyes. The conjunctiva of the left somewhat anæsthetic. The acuity of vision and colour-sense are normal in the right eye. Vision with the left eye is very dim. She requires to bring objects quite close up to her eye to appreciate them at all. Can only recognise Snellen CC. quite close to the eye. The colour-sense in the left eye is unusual. She can only recognise green. All other colours are described as only more or less 'glimmering.' Red in particular glimmers. This test was repeated several times afterwards, with precisely the same result. Usually, according to Charcot and Landolt, green is the next colour after violet which is most readily lost, while yellow and blue may continue to be recognised. Here, however, green only remained—a fact carefully verified by repeated and varied tests.

The extent of the visual field as indicated by the perimeter (Plate II.) taken on June 11, shows that in the left eye the visual field is contracted to an area of about 15 radius, situated somewhat above and to the left of the centre; while that of the right eye, also contracted, has a radius of 50, increasing to nearly 70 upwards and to the left. On July 30, while the

II.



C. Berjeau. Mintern Bro's. imp.

area in the left eye continued almost exactly as before, the right had become contracted to about 30.

A re-examination of this patient on Dec. 3, showed that the symptoms had become rather intensified than the reverse, the patient generally more feeble. The visual fields were seen to be even more contracted than on July 30. The acuity of vision and colour-sense were perfectly normal in the right, while the amblyopia had become still more marked in the left. The only colour recognisable was green, which, however, she now termed blue, instead of green as before.

The disposition of the optic tracts in the chiasma, and their relations to the eyes on the one hand, and their centres on the other, have been the subject of numerous investigations, anatomical, experimental and clinical, the results of which are more or less discordant.[1] On grounds of anatomical and microscopical investigation, Biesiadecki,[2] Mandelstamm,[3] Michel,[4] Schwalbe,[5] support the theory of total decussation of the optic tracts.

Gudden,[6] by tracing the degeneration of the tracts resulting from extirpation of the eyeball, and Nicati,[7] by experimental division of the optic chiasma, support the theory of semi-decussation in all the higher animals; some of the lower, as well as fishes and birds, having complete decussation.

But clinical observation in man places the fact of semi-decussation of the optic tracts beyond all doubt. Many instances of hemiopia from lesions of one optic tract might be quoted. A case reported by Gowers[8] shows this in a very satisfactory manner, and Baumgarten[9] has demonstrated the degeneration of both optic tracts in consequence of destruction of one eye. Cases recorded by Hughlings-Jackson,[10] Remy,[11] and J. L. Prevost,[12] show also that lesions in the region of the corpora geniculata produce a similar result.

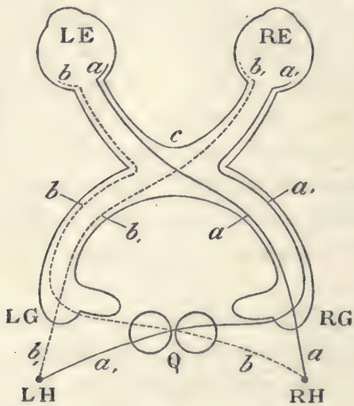
The general disposition of the fibres of the optic tracts,

[1] See Bellouard, 'L'Hémianopsie,' Paris, 1880.
[2] 'Sitzber. d. Wien. Acad.,' Bd. xlii. 1860.
[3] 'Archiv für Ophth.,' Bd. xix. 1873.
[4] *Ibid.*
[5] Von Graefe and Saemisch's Handb. 1874.
[6] 'Archiv für Ophth.,' Bd. xx. 1874.
[7] 'Archiv d. Physiologie,' 1878.
[8] 'Centralb. f. die Med. Wissensch.' 1878.
[9] *Ibid.*
[10] 'Lond. Hosp. Rep.' vol. viii. 1875.
[11] Bull. Soc. Anat. 1875.
[12] Bull. Soc. Suisse, 1879.

apart from the relative position of the decussating and non-decussating fibres, may therefore be regarded as having been satisfactorily determined.

But when we pass from these to the cortical relations, we again meet with difficulties and discrepancies.

Charcot, founding mainly on the facts of cerebral hemi-anæsthesia, considers, as will be seen by reference to his scheme, that the decussation in the chiasma is again reversed,



L E, and R E, left and right eyes; C, chiasma; L G, and R G, left and right geniculate bodies; Q, corpora quadrigemina; L H, and R H, left and right centres of vision; *b* and *a* nerve-fibres from left and right sides respectively of left eye; b^1 and a^1, corresponding fibres from right eye.

probably in the corpora quadrigemina; so that by this the whole of the fibres of one eye are brought into relation with the opposite cerebral hemisphere. Hence the crossed amblyopia characteristic of cerebral hemianæsthesia.

How well this scheme suffices to account for the amblyopia of cerebral hemianæsthesia will be at once apparent, and the facts which I observed in monkeys after destruction of the angular gyrus on one side led me to consider this scheme as exclusively correct. But the recent researches of my colleague Prof. Gerald Yeo and myself—communicated to the Physiological Section of the Brit. Med. Association, 1880[1]—as well as recent clinical observations, render necessary some modifi-

[1] See Abstract in 'BRAIN,' No. XI. p. 419.

cation of this, as well as other previously formulated views on the relations of the eyes to the cortical centres.

First, with respect to the negative or latent effects of lesions of the visual centres in man, viz. the angular gyri (with supramarginal lobules) and the occipital lobes.

We have found in monkeys that the only lesion which is capable of producing complete and permanent loss of vision —if even yet we are warranted in saying so much—is total destruction of the angular gyri and occipital lobes on both sides.

We have found that the effects of all other lesions are more or less transient, and that an animal can see with both eyes if only one angular gyrus remains; and still more remarkable that one occipital lobe is sufficient for this purpose. When we say that the animal sees, we do not merely mean that the eyes are sensitive to light, but that the animal can direct all its actions, and interpret, and act on, its visual sensations in an intelligent manner. We do not pretend to be able, from a study of even the most human of the lower animals, to exclude any affection of vision in its intellectual aspects; but we cannot find that the animal's modes of activity and habits are appreciably altered.

This extraordinary result will, if we may assume a complete physiological as well as anatomical homology between man and the monkey, serve to explain the absence of any permanent loss of vision in connection with lesions which do not destroy the whole area of the visual centres. I do not know of any such case in clinical records, and an event of this kind is not likely to occur under conditions in which it would be possible to arrive at really trustworthy conclusions, on account of the profound impairment of all the cerebral functions which would almost certainly be present.

The effects of unilateral destruction of the angular gyrus speedily disappear in the monkey. Hence in man, especially as the lesions of disease are more commonly only partial, or slowly progressive, the absence of symptoms in the sphere of vision occasions no great difficulty. Bilateral destruction of the occipital lobes (taking care not to trench on the parieto-occipital fissure and the posterior limb of the angular gyrus)

we have found, as I did formerly, to be without discoverable effect on vision. Hence the absence of visual defects in the cases of unilateral and bilateral occipital lesions which have been reported in man[1] is also in harmony with our experimental results.

Next as to the temporary effects of lesions of the visual centres in the monkey.

We have observed, in confirmation of my previously published results, a temporary total loss of vision in the opposite eye when the angular gyrus was completely destroyed. The total loss of vision, however, lasts at most only a few hours, after which period the modes of action of the animal undoubtedly indicate vision, to such an extent at least as to guide its movements and enable it to lay its hand on articles of food. That a condition of amblyopia may exist, however, is possible, though the determination of such a defect is in the monkey practically impossible. And the facts of hemianæsthesia show that there may be considerable amblyopia and contraction of the visual field without the patient being specially aware of it or complaining of defect of vision (see Case II.). If the lesion of the angular gyrus is not complete, the affection of vision of the opposite eye is less marked and more transient. These facts might be taken to indicate that central vision at least is possible, notwithstanding the removal of the opposite angular gyrus.

Recovery is not explicable by the activity of the other angular gyrus only, for we found that the removal of this also, after an interval of some weeks, produced only transient effect. It is different, however, when both angular gyri are destroyed simultaneously. Total blindness ensues; but on the third day there are indications of vision, though as long as three or four weeks afterwards there is still some visual defect, evidenced by the uncertainty and want of precision of aim when the animal tries to pick up articles of food lying on the floor. Extensive lesions, or even bodily removal of the greater portion of one or even both occipital lobes, cause no discoverable impairment of vision.

If, however, the angular gyrus and occipital lobe are

[1] 'Localisation of Cerebral Disease,' p. 116.

destroyed together on one side, vision is distinctly affected in both eyes. A condition of hemiopia ensues; vision being defective or abolished towards the side opposite the lesion, from paralysis of both retinæ on the corresponding side. Whether this hemiopia is symmetrical, or whether each retina is paralysed precisely on one half, or whether one eye is affected more than the other, is very difficult to determine accurately; but we should not pronounce definitively on this point. This condition of hemiopia is also only of temporary duration, though it lasts longer than the cross amaurosis from destruction of the angular gyrus. At the end of a week there is evidence of vision to both sides, and the animal soon recovers, to all appearance perfectly.

From these facts it would appear, therefore, that there is a twofold relation between the eyes and the cortical visual centres; the one mainly cross—the central portion of the retina probably bilaterally represented—by the angular gyrus; the other bilateral—the corresponding side of both retinæ being represented—by the occipital lobe, not alone, however, but in conjunction with the angular gyrus. Perfect vision is possible with the angular gyri when the occipital lobes have been removed almost up to the parieto-occipital fissure, directly after the operation. Perfect vision is also possible by the occipital lobes alone at a relatively short period after the extirpation of the angular gyri; and not only so, but even one angular gyrus, or one occipital lobe may suffice.

Many important questions as to the theory of vision and the comparative relations and development of the homologues of the angular gyri and occipital lobes in different animals are suggested by these results; but at present some of the clinical bearings only will be considered.

The cross amaurosis or amblyopia which we have found to result from lesion of the angular gyrus is entirely in harmony with the affection of vision which is usually seen in cerebral hemianæsthesia. It would therefore appear that the fibres which pass to the cortex in the posterior third of the internal capsule (posterior segment) are those which are specially in relation with the angular gyrus, and represent the opposite

eye as regards those functions which are affected in hemianæsthesia.

That a cross affection of the vision may occur from cerebral lesion seems therefore established both experimentally and on clinical grounds. Nothnagel also[1] regards this as a necessary corollary from the cases already on record. But in addition to—and by some considered incompatible with—this cross affection, the occurrence of hemiopia from cerebral lesion, especially in relation with lesions involving, if not confined to, the occipital lobe and its medullary connections, has been of late strongly urged on grounds of clinical and pathological observation[2] in combination with the physiological experiments of Munk. The cases, however, which have been so far recorded, are, taken by themselves, not sufficiently definite to establish an indisputable causal relationship between the hemiopia and the cerebral lesion as such, or to indicate with any degree of precision the locality of the centre in question. The lesions have either been multiple,[3] or of the nature of tumour,[4] or if of the nature of softening, the lesion has generally been so diffuse and extended so deeply into the medullary substance, that great caution requires to be exercised in founding conclusions on them. This is all the more necessary, inasmuch as lesion or pressure on the optic tract alone causes hemiopia, and this may be affected indirectly though the lesion is not directly situated on its course. The cases also already alluded to, reported by Hughlings-Jackson, Remy, and Prevost, show that lesions in the posterior aspect of the optic thalamus and corpora geniculata cause hemiopia. Hence the possibility of affections of these structures, directly or indirectly, must be excluded before we can consider the causal relationship between hemiopia, and the cortical or subcortical lesions is at all satisfactorily established.

Taken, however, with the facts of experiment above described, the clinical cases have a much greater value. The

[1] 'Topische Diagnostik des Gehekrankheiten,' p. 499.

[2] See Bellouard, op. cit.; and Nothnagel, op. cit.

[3] Cases by Hjort, Förster, Hosch, Chaillon (cited by Bellouard, op. cit.), Huguenin (cited by Nothnagel)

[4] Cases by Pooley, Hirschberg, Jastrowitz, Mohr (op. cit.).

following cases may be selected as the least complicated examples of hemiopia from cerebral lesions.

Wernicke's case [1] was one of right hemiopia, aphasia, and subsequent hemiplegia. There was a cortical lesion of the left hemisphere involving the angular gyrus, a considerable extent of the occipital lobe, and the upper portions of the sphenoid-temporal convolutions as far as the fissure of Sylvius. The lesion extended into the medullary fibres of the ascending parietal convolution, and deeply into the medullary substance of the occipital lobe, dividing the radiations of the optic tract. There was also a focus of softening in the left corpus striatum, but the optic thalami, corpora geniculata and quadrigemina, optic tracts and the rest of the hemisphere presented no abnormal appearance.

Baumgarten's case [2] of left hemiopia had among other slight lesions (one in the roof of the anterior horn of the left ventricle, another in the right optic thalamus), an extensive apoplectic cyst in the right occipital lobe. The rest of the brain was described as normal.

Curschmann's case [3] was one of left hemiopia, occurring without other cerebral symptoms, in the course of fatal poisoning by sulphuric acid. After death there was found "a large softening in the right occipital lobe reaching to the surface, chiefly towards the posterior and inner aspect." The optic tracts and rest of the brain were described as normal.

This appears to be the least uncomplicated case as yet on record, so far as I have been able to discover.

If we may accept these cases as being purely subcortical, and exclude all implication, direct or indirect, of the corpora geniculata and optic tracts, we may interpret them according to what has preceded, as being destructive of the radiations of the optic tract, not merely to the occipital convolutions but also to the angular gyrus. The extent of damage, even though it may appear that the occipital lobe—and this term is used as a rule too vaguely—alone is the seat of lesion, can rarely be determined accurately from the lesions of disease only.

[1] 'Verhandl. d. Physiolog. Gesellsch. zu Berlin,' April 12, 1878.
[2] 'Centralbl. f. die Med. Wissensch.,' No. 21, 1878.
[3] 'Centralbl. f. pr. Augenheilkunde,' June, 1879.

Some interesting cases are also on record of hemiopia resulting from traumatic injuries, more especially in the occipital region.

In one of these reported by Cohn,[1] in consequence of a fall on the head there was right hemiopia and some degree of aphasia for three months after the injury. Several months later, however, both the hemiopia and the aphasia had completely disappeared.

In others, again, the hemiopia has been apparently permanent. The most uncomplicated case of this kind perhaps is the one described by Keen and Thomson,[2] and also by Dr. Otis in vol. i. p. 206 of the 'Medical and Surgical History of the War of the Rebellion,' with a chromo-lithographic illustration of the seat and extent of the injury.

Patrick Hughes received a gunshot wound of the head at the battle of Antietam on September 17, 1862. The descriptions of the course of the bullet and extent of the injury given by different surgeons differ somewhat, but the description given by Keen and Thomson, with which the chromo-lithograph appears to agree, is as follows: "Wound of entrance in the middle line, 1¼ inch above external occipital protuberance—small depressed wound; wound of exit 2 by 2½ inches, its centre being 2 inches to the left of the middle line, and 3 inches above the wound of entrance."

The man was paralysed in the right arm and leg for some time, and considerable fungus cerebri formed and was removed. He, however, recovered within a year, and was able to work. At the date of examination (Dec. 20, 1870), by Drs. Keen and Thomson, well-marked right hemiopia existed, without any pathological appearance in the fundus oculi.

This case is a valuable one, both on account of the nature of the injury and the care with which it has been investigated.

The seat of lesion and the direction of the projectile render it probable that the damage was confined to the posterior lobe of the left hemisphere, touching on, but not permanently destroying the centres for the arm and leg, situated somewhat in advance at the upper extremity of the fissure of Rolando.

[1] Cited by Bellouard, op. cit.

[2] 'Trans. of the Americ. Ophth. Soc.' July 1871, p. 122.

In many cases of injury to the head it is much less easy to exclude direct implication of the optic tract at the base, and this must always be regarded as a possible occurrence, even though the injury might appear to be purely cerebral.

Thus it is highly probable that pressure on the optic tract is the cause of the temporary hemiopia described by Gowers[1] in connection with apoplectic extravasations, existing along with conjugate deviation of the eyes. The hemiopia is towards the side opposite the lesion. We may suppose that there is, for the time being, pressure on the optic tract on the side of the effusion. This at least would seem to be the cause, unless it can be shown that other forms of sensibility, general and special, on the opposite side are also for the time abolished; facts which would indicate rather temporary annihilation of the functions of the whole hemisphere.

That a cerebral lesion in the occipito-angular region may cause hemiopia altogether apart from direct or indirect implication of the optic tract or corpora geniculata is, however, possible, is rendered clear by the experimental facts above recorded, and some of the clinical cases are best explained on this hypothesis.

A temporary functional disturbance of this region appears also most satisfactorily to account for the scintillant scotoma, so frequent in connection with migraine, the phenomena of which have been minutely described by Airy,[2] Liveing,[3] Dianoux,[4] and others.

Sometimes only one eye is affected, wholly or partially, at other times a fugacious homonymous hemiopia is observed.

In addition to the ocular symptoms, there are not unfrequently other paræsthesiæ and parakineses on the hemiopic side, with temporary disturbances of speech of the aphasic type. These phenomena, with the accompanying acute hemicrania, may all be accounted for by neurosis of the cerebral membranes, and temporary disturbance of the functions of the subjacent cortical centres.

If we consider it sufficiently well established by the above

[1] 'Brit. Med. Journal,' Nov. 24, 1877.
[2] Proc. Roy. Soc. Feb. 1870.
[3] Megrim, 'Sick Headache,' &c., 1873.
[4] 'Du Scotome Scintillant,' 1875.

facts and considerations that hemiopia may be caused either by lesion of the optic tract, or from cortical or subcortical lesions in the occipito-angular region, it becomes an important question whether we can distinguish during life between the one and the other.

On this point we are naturally considerably aided by the simultaneous occurrence of symptoms indicative of a cerebral origin, such as hemiplegia, hemianæsthesia, aphasia, &c., but these alone are not altogether sufficient, nor are they always present, nor would they exclude simultaneous implication of the optic tract by pressure or otherwise.

A perimetric investigation of the eyes in hemiopia, proved to be due to direct lesion of the optic tract[1] shows the visual field divided by an almost vertical line passing through the point of fixation. On the one side of this line vision is entirely abolished to the right or left, as the case may be, both centrally and peripherically. Each optic tract supplies precisely the corresponding half of both retinæ.

On the other hand, there are many cases of homonymous lateral hemiopia, in which, though the lateral defect has been of indefinite duration, central vision is retained in both eyes for some distance on all sides of the point of fixation.

This is a fact of great importance and signification, and one to which I would direct especial attention as likely to furnish a means of diagnosis between cerebral and peripheral hemiopia.

In cases where central vision is retained for some degrees on all sides of the point of fixation I should regard the cause of the hemiopia as cerebral. The facts above recorded show that the angular gyrus of each hemisphere has a bilateral relation with the centre of the retina, so that, notwithstanding the complete destruction of the visual centre in one hemisphere, central vision is possible with both eyes, through the sound angular gyrus. Hemiopia therefore may exist towards the side opposite the lesion, with retention of central vision in both eyes.

The facts both of experiment and of cerebral hemianæsthesia, indicate that the area of central vision is more exten-

[1] Gowers' case, supra cit.

sive on the eye opposite the sound side; but central vision, more or less, is still possible though the opposite centre has been destroyed.

A rigid perimetric investigation is therefore a matter of great importance in reference to this question, and a careful comparison of the relative extent of the area of central vision in each eye desirable.

In many of the cases of hemiopia supposed to be due to injury or disease of the occipito-angular region, the perimetric investigation has not been recorded in such a way as to settle this point.

But in the case of Patrick Hughes,[1] Drs. Keen and Thomson found that in the right eye—the hemiopic side —"at the point of fixation the vertical line which bisected the field seemed to deviate slightly towards the defective side."[2] The figure they give shows a distinct area of central vision, interrupting the vertical line above and below the point of fixation.

Though they suggest excentric fixation and the possibility of the patient having followed the light inadvertently, yet the facts are more in accordance with the explanation I have suggested, viz. the functional activity of the sound angular gyrus.

Hirschberg regards it as the rule that in homonymous hemiopia the vertical line always diverges towards the defective side near the point of fixation, so as to have an area of central vision ranging from 3° to 5°. Where this is so, we may regard the hemiopia as of cerebral origin. And I am of opinion that careful examination will in all cases of cerebral hemiopia establish a greater clearness of central vision and over a larger area, in the eye on the side of lesion than in the opposite. An examination of some of the cases with the perimeters given by Schweigger,[3] also bears this out. The point, however, is one which I must leave to ophthalmologists.

It is a question whether there is any difference between cerebral and peripheral hemiopia, in respect to the degree and time of occurrence of atrophic changes in the optic disc.

[1] Op. cit. p. 126.

[2] 'Archiv f. Ophthal.,' Bd. xxii. Abtheil. 3, 1875.

[3] See page 470.

The statements as to the occurrence of atrophy in hemiopia are somewhat at variance. In many cases, even of long duration, ophthalmoscopic examination seems to have been negative. Gowers[1] has uniformly failed to discover it. Others again have described it, but while some have seen atrophic changes in both papillæ ; others again, as Mauthner, state that in homonymous hemiopia atrophic changes are visible only in the disc on the hemiopic side. In a case of right hemiopia, however, of over ten years duration, which I have carefully examined in reference to this point, I have found atrophy of both discs, but certainly more pronounced on the right.

It is certain, however, that atrophy of the optic nerve does not occur so readily as atrophy in cases of lesion of peripheral nerves in general, a fact which may be due, as Gowers suggests, to the so-called trophic influence of the retina itself.

And this is supported by the fact that also in heteronymous, temporal or nasal, hemiopia, depending without doubt on peripheral lesion, the ophthalmoscopic examination is often, if not constantly, negative.

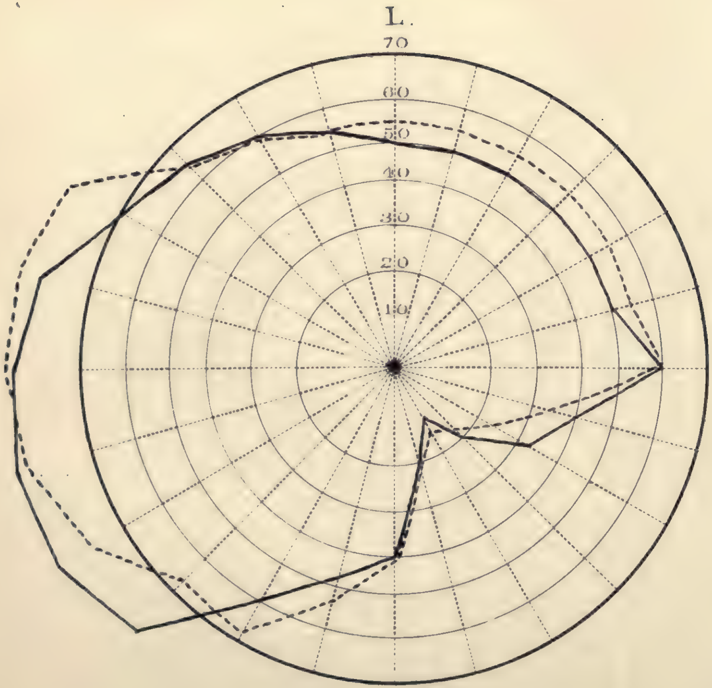
The question is one, however, which still requires investigation. That pale atrophy of the optic discs may occur from purely cerebral lesions altogether apart from lesion of the optic tracts, is a fact which the experiments of Dr. Yeo and myself—to be published in detail at a future date—clearly indicate.

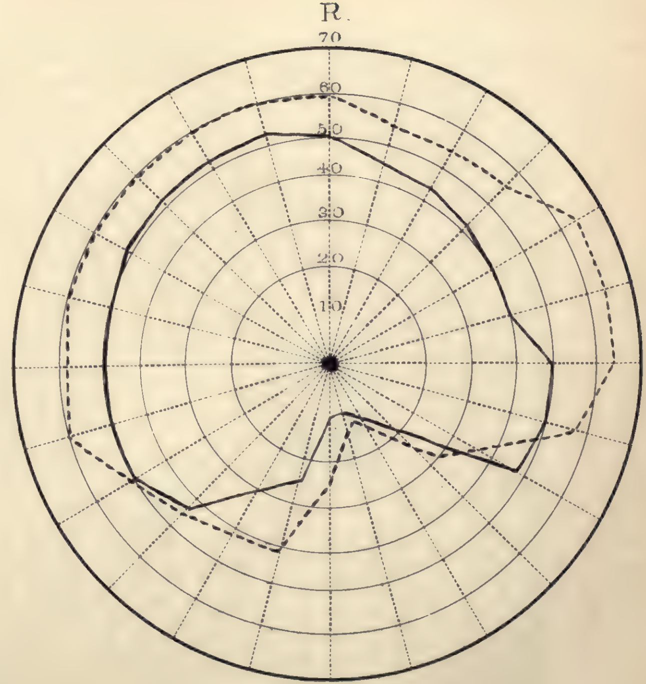
The mere fact of the occurrence of optic atrophy would not therefore appear to constitute a means of diagnosing cerebral from peripheral hemiopia, but we might say that a rapidly occurring and decided atrophy of the optic discs would indicate peripheral rather than cerebral hemiopia, as direct lesions of the optic tract are more likely to be propagated to the retinal expansion of the optic nerve.

As to the duration of hemiopia depending on cerebral lesion, if we might argue without qualification from the monkey to man, we might expect that it should in time clear up and disappear. But the facts relating to cerebral amblyopia in connection with lesion of the posterior segment of the internal capsule seem to show that the duration of visual

[1] 'Brit. Med. Jour.' Nov. 1877.

III.





C. Bergeau

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defects in man is much greater than in the monkey. Persistent defect in the acuity of visual perception and discrimination can be demonstrated in man, long after any defect as to mere light and shade, on which we have mainly to rely, can be recognised in the monkey's behaviour and modes of activity. It is further quite possible that the recovery from hemiopia in this animal may not amount to fine visual discrimination, though the perception of light may be sufficiently restored for ordinary modes of activity. The higher specialisation of the visual sense and important intellectual relations of binocular vision in man would lead us to believe that recovery after total destruction of the visual centres in one hemisphere would rarely if ever be absolutely perfect. The question is whether recovery to some degree at least may occur. The prognosis in cerebral hemiopia is generally held to be unfavourable. The hemiopic defect existed in Keen and Thomson's case (p. 470) eight years after the infliction of the injury to the brain.

In a case recently under my observation, however, of which the following is a condensed abstract of the notes, the symptoms as to vision were such as indicate gradual recovery from an extensive damage of the visual centres, and perhaps may throw light on certain forms of scotomata sometimes met with, the pathology of which is still obscure.

Case III.—J. S., ætat. 43, admitted into the National Hospital for Paralysis and Epilepsy on March 5th, 1880. Had an apoplectic seizure, with more or less unconsciousness for several days in Aug. 1879. On regaining consciousness, found that he was paralysed as to sensation and motion on the right side, with some degree of aphasia. He observed at this time that he could only see the left half of objects.

At the time of admission he was still suffering from considerable right hemiparesis and right hemianæsthesia, both as to tactile and muscular sensibility, hearing, and taste. Smell, however, was abolished in the left nostril instead of the right as usual.

The note of the examination of the eyes simple states that right hemiopia existed, but unfortunately no exact perimetric examination was made at this date.

The perimeter taken on 23rd March (Plate III.) shows a

want of symmetry in the visual fields, the right being considerably smaller than the left. At the same time there is in both a symmetrical quadrant-shaped scotoma downwards and to the right. The areas of colour-vision were found to correspond to the normal. On May 21, after two months' treatment, considerable improvement had occurred both as to motion and sensation on the right side.

The perimeter at this date shows that the visual field has become enlarged correspondingly in both eyes, the want of symmetry being retained.

The scotoma downwards and to the right still continues.

The ophthalmic appearances were negative.

The other symptoms in this case and the want of symmetry in the visual fields all indicate a cerebral origin. If we suppose, though unfortunately it cannot be demonstrated, that there was true homonymous lateral hemiopia in this case—and the patient was very positive as not having been able to see to the right either upwards or downwards at first—this might be looked upon as a partial recovery from cerebral hemiopia, and defects of this kind may be regarded as the remnants of a hemiopia at first complete. I find that a similar case in which complete recovery ensued has been recorded by Lang.[1] But the explanation of this and similar cases which most readily occurs is that the cerebral lesion has been only partial, and that, as in aphasia, the recovery is not so much the compensatory activity of the centres in the other hemisphere as the restoration, more or less complete, of the centres which have been damaged.

I have not been able to find any cases on record of hemiopia undoubtedly of cerebral origin in which the centres have been proved to be completely destroyed in one hemisphere, followed by recovery, unless we can regard the cases of latent occipital lesions as such. On referring to the recently published lectures by Mauthner,[2] which I have seen just before sending these pages to press, I find that he regards the prognosis of true homonymous hemiopia as almost invariably unfavourable, and that if it has existed for a few weeks a

[1] 'Centralbl. f. pr. Augenheilkunde,' July 1880.

[2] 'Vorträge aus dem Gesammtgeb. der Augenheilkunde,' Heft. 6–8. 1881.

restitutio ad integrum is not to be looked for as at all probable (p. 371).

He however records a case of right hemiopia (p. 365) of thirteen years' duration at the time of the first examination, which four years subsequently, i.e. therefore seventeen years after its occurrence, had recovered so far that on the right half of the fields formerly quite dark, not merely was light perceived, but a considerable amount of visual perception had returned. Colour perception remained quite abolished, however.

It is doubtful whether this was a case of true cerebral hemiopia, however, as visual defect in both fields was said to have reached quite up to the fixation point.

But it is a point worthy of future investigation, even in long-standing and apparently incurable hemiopia of cerebral origin, whether indications may not be discoverable of gradual improvement by the compensatory action of the sound visual centres, and whether this may not be fostered and stimulated.

DESCRIPTION OF PLATES.

PLATE I.—Field of Vision, R(ight) and L(eft), of Thomas H. (Case I., p. 459.)
The continuous line indicates the extent of the field of vision on April 24.
The dotted „ „ „ „ June 5.

PLATE II.—Field of Vision, Right and Left, of Hannah O. (Case II., p. 461.)
The continuous line indicates the extent of the field of vision on June 11.
The dotted „ „ „ „ July 30.

PLATE III.—Field of Vision, Right and Left, of James S. (Case III., p. 475.)
The continuous line indicates the extent of the field of vision on March 23.
The dotted „ „ „ „ May 21.

VISIBLE MUSCULAR CONDITIONS AS EXPRESSIVE OF STATES OF THE BRAIN AND NERVE CENTRES.

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COARSE or extensive paralyses such as hemiplegia, paraplegia and other profound disturbances of the muscular system have received much attention from clinical and pathological observers, and by the accumulation of their joint observations much knowledge has been gained as to the symptoms that result from lesion of certain portions of the brain. This should encourage us to observe in all cases the conditions of the muscles, knowing that the movements correspond to the states of certain nerve-centres, and looking upon such nerve-muscular conditions as indications or expressions of the states of those centres.

Epilepsies has been closely observed and accurately described, with the result of adding to our knowledge of brain conditions. Chorea, paralysis agitans, and the movements seen in athetosis, and disseminated sclerosis of the cord, were described as nerve-muscular conditions, and were empirically observed before their pathology was discovered. In these cases by studying the palsies, spasms, tremors, rhythmical and other forms of muscular action, we are enabled to gain some idea of the state of different portions of the brain and spinal cord. All expression of feeling is effected by muscular action, whether it be by words, by facial movement or gesture, movements effected by voluntary muscles; or expression may be produced by dilatation of the pupil, erection of the hair,

or disturbed action of the heart, these being due to the conditions of inorganic muscular fibres.

For these reasons I have been accustomed to look rather closely at the nerve-muscular condition of "nervous cases" when seeking definite signs by which to describe them, sure of the axiom (with certain exceptions, such as conditions of muscular irritability, and muscular disease) that movements depend upon nerve-muscular stimulus originating in nerve-centres.

Examples may easily be given showing how we commonly judge of the state of the nervous system by muscular conditions. Note the stooping attitude and spiritless gait of a tired man as compared with that of the same individual when rested and refreshed. Incipient intoxication is indicated by a reeling gait, unsteady hand, and muscular tremor. Expression may be indicated by the position of the head, it is seen firmly upright in defiance, drooping in shame; it is commonly held on one side in nervous women, and girls convalescent from chorea, the first example cited of an asymmetrical gesture. The artist's brush or pencil, the sculptor's modelling tool and chisel, the pianist's and violinist's finger-touch, indicate the training and actual condition of the working of his brain. The educated and refined singer trains and refines his whole mind, i. e. his brain, and is well aware that his "whole soul" as he may express it, comes out in the action of the muscles concerned in producing his song, and musical notes.

In the infant the condition of the nervous system is best recorded in terms of nerve-muscular phenomena. It laughs and is playful; reflex action is well marked when a finger is placed in the child's hand, or mouth. The eyes are moved and directed towards any object looked at; these are conditions of healthy action. It is well known that in the convulsive state the fists are often closed with the thumbs turned in.

All these examples of expression are nerve-muscular conditions; the movement, the attitude, the gait result from states of the brain or spinal cord.

In the observations to be referred to, examples are chiefly drawn from the ocular and facial muscles, and those of the upper extremity. Muscular movements in the eyeball are

seen in the varying conditions of the pupil under the influence of light, and in the changes accompanying accommodation for near and distant vision.

In the iris we have a very sensitive and beautifully adjusted muscular mechanism, in connection with the third nerve of the brain and the sympathetic. The clinical study of this muscular organ and its indications were fully and carefully treated by Mr. Hutchinson in a paper in 'BRAIN,' Vol. I.

Movements produced by muscles outside the eye are seen in the act of directing the eyes in any given direction, these movements being so governed that the parallelism of the axes of the eyes is usually maintained; these co-ordinated movements being probably governed by some brain centre. In looking at objects about two inches from the face, these recti muscles outside the eyeball cause convergence of the axes of the eyeballs. To allow of such convergence, the simultaneous and equal contraction of the internal rectus muscle of each eye must be accompanied by the relaxation of the external recti; this complicated association of muscular conditions must be regulated by the nerve-centres. These movements are the normal.

We now pass on to consider departures from these normal conditions. I have shown that in deep anæsthesia from chloroform,[1] or in coma from alcoholism, or in the profound sleep of infants, the loss of associated movements of the eyes may be complete. If in an adult deeply under chloroform, the eyelids be gently raised, the pupils will be seen minutely contracted, often to a pin-point, the eyes having at the same time lost the parallelism of their axes. One eye may move upwards or outwards while the other remains quiet, or moves in a different direction, or at a different pace, thus causing a temporary and varying strabismus. Usually these movements are confined to the horizontal plane, less commonly the eyes assume a different level, one being in the horizontal plane, while the other is turned downwards. It is noteworthy that the average continuance of the eyes in the horizontal plane of the axes of the orbits, is in accordance with other examples

[1] See paper, 'Brit. Med. Journal,' March 10th, 1877.

of involuntary movements of the eyes, to be presently noted, e.g. nystagmus, and irregular jerking movements of the eyeballs. These movements I have frequently observed in the healthy subject, and seen that though they occurred thus irregularly while in coma, the action of the pupils and the co-ordination of the eye-movements was completely restored on recovering consciousness. In the profound sleep of an infant in its mother's arms the same loss of association of movements occurs, but at the moment of waking the pupils dilate, and co-ordination of movements is restored; the child must be profoundly asleep to allow of the eyelids being raised without awaking it.

Case I.—At the Children's Hospital, Birmingham, in 1876, I saw a girl three years of age the subject of permanent hemiplegic paralysis, following convulsions in infancy. She had never spoken, was constantly dribbling, and idiotic in manner. There was an occasional loss of the parallelism of the eyes; one would remain at rest, while the other wandered inwards or outwards; this was a chronic condition in the child, who was suffering from no acute disease. The symptom appeared due to the defective condition of the brain.

Case II.—Through the kindness of my friend Dr. Fletcher Beach, I had the opportunity in 1877 of seeing in the Clapton Asylum a microcephalic idiot of very low development, in whom the eyes almost habitually wandered about independently of one another. In very weakly infants, not the known subjects of brain disease, the loss of associated movements may be very distinctly seen at times while awake and sucking at a bottle. In cases of meningitis, and other conditions of coarse brain disease, the same condition is sometimes seen.

Lastly, it is interesting to call to mind that in many of the lower animals there is the power of moving either eye separately and independently, so that that centre which co-ordinates the movements of the two eyes may be looked upon as a "later centre," i.e. one more recently developed in the ascent of Man. Probably some interesting and important points concerning the movements of the eyes might be elucidated by considering them in connection with those of the lower vertebrata.

Nystagmus is a condition producing a rapid oscillation of the eyeballs; usually the parallelism of the axes of the eyes is maintained, and the movements are in most cases in the horizontal plane of the axes of the orbits. Occasionally the movements are vertical, as in the cases of two brothers with retinitis pigmentosa, frequently seen in the clinique at Moorfields about 1870.

These cases of vertical nystagmus are uncommon, so also I believe, it is unusual to have one eye only thus affected.

Case III.—Emily B., aged 4 years, was sister to an infant under my care at the East London Children's Hospital for congenital syphilis. When seen, nystagmus of the right eye was at once observed. There was inconstant vertical oscillation of the right eyeball, without any lateral movement; the movement was seen when the child looked at distant objects, not when looking at near objects; there was no nystagmus of the left eye. The pupils were equal; there was no strabismus. The normal movement of the eyes could be performed, together with the natural convergence for near vision. Ophthalmoscopic examination was impossible, on account of photophobia. There was good vision in both eyes; the child was intelligent.

Case IV.—In an infant which I had the opportunity of seeing at the London Hospital while she was under the care of Mr. Hutchinson, there was congenital nystagmus of both eyes; the rapidity of movement in the two eyes varied greatly, but it was impossible to count the oscillations. This would appear to indicate that the nerve-centres ruling either eye and regulating the tension of one rectus muscle when its opponent is relaxed, act more independently than in the normally developed brain, i. e. the centre whose office it is to co-ordinate the associated movements of the two eyeballs is feeble or absent in such a case, as it appears absent in some idiots, and paralysed (in healthy brains) in deep coma.

Case V.—William D. was the subject of right hemiplegia and presented syphilitic lesions in the soft palate, pharynx and bones, and there was atrophy of the right half of the tongue.

There was no strabismus, and he could move the eyes in any direction; when the eyes were directed in an extreme

degree to either side horizontal oscillatory movements resulted, the lateral jerks reminding one of the more rapid oscillations of nystagmus, but these movements only occurred when the eyes were directed towards the right or left hand; no such oscillations resulted on looking upwards or downwards. Another noteworthy phenomenon concerning the muscles of the eyes is the fact of convergence for near vision: an additional example of movements in the horizontal plane of the axis of the orbits. This movement is well seen in a healthy individual by causing him to look at a distant object and observing the size of his pupils and the parallelism of his eyes, then causing him to look at his nose the eyes are seen to converge, directing their axes towards the object, at the same time the pupils contract.

Case VI.—Mr. Priestley Smith, in the Ophthalmic Hospital Reports, vol. viii. part ii. narrates the case of a lad in whom the lateral movements of the eyes were impaired. "Both eyes were fixedly turned towards the patient's right. The deviation, measured by prisms, equalled twenty degrees in each when looking at a distance. The head was kept turned towards the left to counteract the position of the eyes. An object was moved from right to left, and left to right in front of the patient. Neither eye could follow it to the smallest extent towards the left, but both, on the contrary, were capable of a very slight additional turning towards the right. This they performed, however, in a jerking manner, and at once returned to their former position. An object was then held at ten feet distance and rather towards the patient's right, so that his eyes could be fixed on it, and it was then made to approach his face. Both eyes steadily converged so as to remain fixed on the object until it was only five inches from the face. This involved a remarkable phenomenon, viz. the right eye, which had before refused to move towards the left, now made a considerable excursion (about fifteen degrees) in that direction." In this highly interesting case collateral deviation of the eyes was marked, the power of convergence for near objects being retained.

Ferrier has shown[1] that there exists in the convolutions of

[1] 'Functions of the Brain,' p. 229.

the frontal region a brain centre, which when excited causes both eyes to turn away from the side excited; if on the contrary this centre be destroyed instead of excited, the corresponding centre in the other half of the brain, acting unopposed, turns both eyes towards the side of lesion. Such a lateral deviation of the eyes, parallelism of their axes being maintained, is a common condition in cases of hemispasm and in recent hemiplegia.

In health the greater number of ordinary movements of the eyes are probably in the horizontal plane of the axes of the orbits. Thus the movements of accommodation for near vision are in this plane, in nystagmus the oscillations are usually so, also the jerking movements in the case V., and the involuntary movements with loss of parallelism seen in coma, are mostly horizontal. The facts point to the horizontal movements as the commonest, the least voluntary, and probably the least intellectual. The horizontal movements of the eyeballs do not involve movements of the eyelids by muscular action, i. e., the horizontal movements of the eyeballs do not cause simultaneous movements of the muscles of the eyelids, therefore I think horizontal movements the least intellectual. To turn to vertical movements. Dr. Gowers has shown * that the movements of the lower eyelid constitute a simpler problem than those of the upper lid. The lower lid follows the movements of the eyeball upwards and downwards, but not very closely. No muscular mechanism exists which can cause the downward movements of the lower lid; such movement is produced by the movement of the eyeball acting mechanically upon the lower lid. The upper lid possesses a more complex mechanism. The descent of the lower lid on downward rotation of the globe is not due to the contraction of the orbicularis. Simultaneous with the descent of the upper lid in the downward movement of the eyeball, there must be a relaxation of the levator. In upward rotation of the eyeball, contraction of the levator, is associated with that of the superior rectus. The association of the levator and superior rectus suggests that both are relaxed or energised in similar degree when the eyeball is moved upwards or downwards; in

* 'Med. Chir. Trans.' vols. i. and ii.

accordance with the law, which it is evident must obtain in all muscular actions, that the opponents of the muscles producing the movement, are relaxed in exact proportion to the degree of movement produced. Thus in the upward vertical movements of the eyeballs, a more complex movement and especial muscles come into play.

Contrasting the horizontal with the vertical movements, we see that the former involve only movements of the rcecti muscles. Of movements of the eyeball in the vertical plane those in the downward direction involve contraction of the inferior rectus only, but at the same time the levator must be relaxed; this co-existent relaxation of the levator and contraction of the inferior rectus suggest that the downward movements are more intellectual than the horizontal. In upward movements the levator contracts synergically with the superior rectus, and the co-existent activity of the two muscles points to these as probably the most intellectual of all. This is again another point as to which it would be exceedingly interesting to know the teachings of comparative anatomy to see whether this power of upward turning of the eyeball, with elevation of the upper lid, be not a later developed power.

From direct observation it appears to me that intellectuality is represented by the movements of the eyeballs in their orbits. When an individual in looking at an object moves the eyes by the action of the recti muscles so as to direct them towards it, the movement is more intellectual than when the head is turned so as to direct the eyes in the required direction. A bright, healthy well-developed infant turns its eyes well in the orbits in looking about, not so a dull wasted child.

Again, intellectual people usually move the eyes in their orbits in looking at any object; the low and vulgar often move the head in the direction required. Here again is a question as to the meaning of which comparative anatomy might give some light.

In chorea we frequently see that although the eyes never lose their parallelism, they are often jerked about in a perfectly involuntary manner, and clearly without the patient desiring to look at the successive objects towards which the eyes are turned. It would appear that the brain-centre which governs

the co-ordinated movements of the eyes is never paralysed in chorea as in conditions of coma, but that it may be "choreic"; this centre then may be temporarily paralysed in healthy infants, or in healthy adults by chloroform; it may be absent in idiots; in weakly infants the slight disturbance of sucking (cerebral anæmia?) may paralyse for the time this centre; this centre is absent in many lower vertebrata.

In making observations on these associated movements of the eyes we are strongly reminded of Ferrier's experiments* on excitation of different portions of the cerebellum. I quote a few results. "Both eyes turn to the left or right in a horizontal plane"; "both eyes move straight downwards"; "both eyes move upwards and to the left"; "both eyes move upwards," etc. These results of experiments suggest that possibly it is the condition of these cerebellar centres that leads to the eye movements described and referred to.

We now come to the study of visible conditions of the facial muscles in expression of the state of the nerve-centres.

The method I have used in analysing the muscular condition of faces is as follows:—holding a piece of paper with one edge vertical, either half of the face is covered in turn, it is thus seen whether the face is symmetrical. Again the face may be divided into three zones by holding the paper with one margin horizontal, leaving the forehead above the eyebrows uncovered; or the face below the lower margin of the orbit may alone be exposed, showing the mouth, most of the cheeks, and the alæ nasi; or again, the middle zone including the eyelids may be viewed alone.

Most of the more definite forms of expression by muscular action are symmetrical, and the face is no exception to this rule. Asymmetry of the face is not met with in many instances. It is seen in facial palsy, whether from disease of the portio dura of the seventh nerve, or from brain disease; it is also seen in the expression called sneering when a one-sided muscular action partially uncovers the canine tooth; and in winking. Occasionally in very nervous people a one-sided grimace may be seen. Other cases of asymmetry of the face may be seen, but are rare.

[1] Op. cit. p. 98.

Before proceeding to describe symmetrical conditions of the face illustrative of the subject under consideration, it may be well to consider what we see when we look at a face.

1. There is its *form*, outline, and the relative position of parts.

2. *Colour*, a condition of the surface which may be in part due to pigmentation of the skin and other conditions, as well as to the state of its circulation.

3. *Movement* of the parts of the face from tone, action, or relaxation of the muscles. It is with these conditions of the muscles that we are principally occupied.

Certain commonly marked lines in the face exist as the result of the puckerings produced by muscular action. In the frontal zone horizontal lines are produced by the action of the occipito-frontalis, and vertical lines are produced by the action of the corrugators. In the middle zone the most pronounced lines are the naso-labial grooves; we also specially here observe the lines of the upper and lower lips, and the positions of the angles of the mouth. The mouth may be widened, or one or both angles may be drawn upwards and outwards or downwards.

The attempt has been made to determine something of the intellectuality of the different facial muscles. The problem was commenced on the negative side by observing the conditions of the face in fifteen idiots. In conjunction with Dr. Fletcher Beach, of Darenth Asylum, I analysed each face according to the following form :—

General muscular condition . .	The action, or relaxation, of the muscles of the limbs and body generally, were noted.
Face	Facial aspect, and muscles in action or relaxation.
Upper zone . .	Frontal region, occipito-frontalis and corrugator.
Middle zone . .	Eyelids and orbicularis oculi.
Lower zone . . .	Mouth muscles; muscles of nose; cheeks.

Summarising from such forms the muscles most often seen in meaningless action, the following results are obtained :—

Occipito-frontalis	11	Depressor anguli oris . .	5
Zygomas	8	Orbicularis oculi . . .	3
Corrugator supercilii . . .	7	Grief-muscle	2

With regard to the teaching of these figures, so far as these few cases go, it shows the frequency with which these muscles

respectively come into spontaneous action in a meaningless manner. This is perhaps some indication of the degree of their intellectual representation. Thus the grief-muscle and orbicularis oculi were much less frequently thrown into meaningless action than the occipito-frontalis and zygomatic, and probably these former are much more expressive of intellectuality than these latter.

In the above table the number 7 is put against the Corrugator; but this I think needs explanation; for from direct observation in healthy intellectual faces, I think that this muscle is one of the most intellectual. If, instead of putting one against the name of a muscle as it may be seen frequently in action in a particular face, we put one or one half, according as its action is marked or only slightly indicated, on summarising these numbers we get for the occipito-frontalis 10·5, for the corrugator 5·0. This result indicates the slight action of the corrugators in the faces of idiots as compared with the occipito-frontalis.

Again, applying direct observation to the other side of the question, and noting which muscles are most frequently put in action in the faces of intellectual people in the expression of their mental states, I think that we see intellectuality most commonly expressed in the frontal and middle zones, and by the action of the corrugator and orbicularis oculi muscles.

Among out-patients it has frequently been possible from the muscular condition of the frontal region (principally increased tone or contraction of the corrugators), and depression of the angles of the mouth (dep. ang. oris) to accept the facial appearance as a physical sign of the "mental state" of intellectual suffering, and physical or organic suffering. On inquiry in such marked cases some painful condition and some source of anxiety or mental distress has almost invariably been found.

Much has been said[1] at various times about facial expression. I will not add thereto, but suggest that to describe such expressions in anatomical terms is a matter of physiological importance, as giving more exact indications of the nerve-centres.

[1] See Darwin on Expression of the Emotions; Bell on the Face; Camper, 1780; Bulwer, 1649.

Taking the face as a clinical region of observation, special points sometimes observable in the different zones may be described. When specially studying the faces of patients, the subjects of recurrent headaches, and analysing them daily, my attention was particularly drawn to the middle zone.

It is not uncommon to observe that an individual "looks as if he had a headache." Analysing such faces it soon became noticeable that there was a look of depression, heaviness, fulness about the eyes, especially about the under eyelid. It appeared that this expression must be due principally to the condition of the orbicularis palpebrarum. There was obviously no pitting on pressure, no œdema, and when the face is œdematous this relaxed look is not seen. Specially observing the orbicular muscle and the parts adjacent, there seemed to be a loss of tone in it; there was an appearance of fulness and flabbiness; the skin hung too loosely, with an increase in the number of folds; and, in place of falling against the lower eyelid neatly, as a convex surface, it fell more or less in a plane from the ciliary margin to the lower margin of the orbit. This condition is often seen best by looking at the patient's face in profile. It was often seen when the skin was healthy and elastic; and that it is due to the muscle seems demonstrated by the change produced if the patient can be induced to laugh, when the muscle is energised, recovers its tone, tucks in the skin well against the eyeball, and the expression of headache is lost for a time. It is not suggested that this muscular condition indicates only the states producing headache; it appears common in after-states of depression.

It may be remarked that the eyeball is probably almost entirely lacking in expression. If a man wear a mask, showing the eyes only, there is no certain expression. It is the custom in some parts of Italy for men to beg in silence, wearing a loose garb, and hood covering the face, with holes showing the eyeballs only, and the absence of expression is marked.

Doubtless in rage we have the dilated pupil, and it contracts with accommodation for near vision; it may also have a characteristic pigmentation; but, speaking generally, the eyeball almost lacks expression, and the special appearances observed about the eye are due to conditions of the eyelids

and the muscles which move them (the orbicularis and levator palpebræ), and in some degree to the muscles which move the eyeballs.

The orbicularis oculi is not solely supplied by the facial nerve, but has also some supply of nerves from the sympathetic.

It is likewise one of the muscles not greatly paralysed by facial palsy due to brain disease. Indeed in Bell's paralysis the eye can generally be closed.

Case VII.—In a case of congenital Bell's paralysis,[1] from ear disease, in an infant, accompanying hemiplegia on the opposite side, the eyelids on the side of paralysis were closed when the face was blown on. This muscle is much less paralysed than the zygomatic in cerebral facial palsy.

In the lower zone are many points of interest to be noted. With regard to the depression of the angles of the mouth in feelings of sorrow, I have frequently observed that when a mother has just lost her child there is a marked depression as from physical suffering.

Case VIII.—In the case of a mother speaking to me of the loss of her children four years previously, there was no depression of the angles of the mouth, but contraction of the corrugators; this would seem to point to the passing away in the course of years of the expression of mere physical suffering from the loss of her children, and the more permanent expression of intellectual suffering from the bereavement.

Case IX.—A woman came to see me as an out-patient, ill with the effects of distress at the loss of a son, aged 21 years, and married only three months. He had died of small-pox a month previously. Her aspect was emotional, she flushed much, and there was marked expression of distress, indicated principally in the frontal region by action of the corrugators, and slight contraction of the inner portion of the occipito-frontalis. The angles of the mouth were not depressed. The face appeared to indicate mental distress and anxiety rather than mental suffering.

The compressoris alæ nasi under ordinary circumstances and in dyspnœa act rhythmically, and synchronously with the respiratory movements, under the guidance of the respiratory

[1] 'Brain,' Part VIII.

centre. In slowly advancing death the alæ nasi may become paralysed, and so collapse on inspiration. Again, in chorea these muscles are often distinctly choreic, being jerked with the other facial muscles, thus seeming to indicate they receive the choreic impulse from the facial centre, not from the respiratory, as they do not jerk synchronously with the other respiratory muscles.

Among asymmetrical conditions of the face the most marked are due to paralysis. Facial paralysis occurs from two principal causes—(1) lesion of the facial nerve; and (2) lesion of one side of the brain, attended with paralysis of the upper and lower extremity, and of the face on the opposite side. In the case of facial palsy due to brain disease, the muscles about the angle of the mouth are principally weakened. This is illustrated by the photograph of a man the subject of left hemiplegia. If either vertical half of the face be covered, it is evident we have an asymmetrical condition. If then we cover each zone in succession, and again look at each zone separately, we find the asymmetry is in the lower zone about the mouth; it consists in a falling of the left angle of the mouth from weakness of the muscles, which expand the mouth and draw its angle upwards and outwards.

This may be compared with the photograph of a case of Bell's paralysis on the right side, in which all the facial zones are affected in an asymmetrical manner, the muscles all being weakened on the same side.

In connection with the fact that brain disease causing one-sided facial paralysis affects the muscles about the angle of the mouth the most, we are reminded that the zygomatics are among the most frequently active muscles in the movements of the faces of idiots, as recorded in the table. These muscles are also sometimes seen acting very asymmetrically in very nervous people. A very moderate amount of brain disturbance appears to cause irregular action or weakening of these muscles.

Again we are reminded of Ferrier's[1] experiments. I quote the results of the excitation of points in the ascending frontal convolution: "(7) action of the zygomatics, by which the angle of the mouth is retracted and elevated." (8) "Elevation

[1] Op. cit. p. 143.

of the ala of the nose and upper lip, with depression of the lower lip, so as to expose the canine tooth on the opposite side."

Some points in favour of the clinical study of nerve-muscular conditions of the face are illustrated by the passive, expressionless face which may be woke up, "lighted up," made to express "the whole soul in the face" simply by conditions of tension of the facial muscles resulting from the mental state. Thus often great and most pleasing beauty is seen in faces unattractive when at rest. Conversely some faces are beautiful in their passive condition, but lack expression and interest when in action from mental work; such women talk but little. Surely from these two considerations it is suggested that the passive form and colour of a face are qualities not as great, not as much mind-indicating, as the mobile expressions produced by muscular tension.

Possibly we may for a moment be allowed to step out of our groove and see how education, and thoughts and habits of thought, and feeling, implanted in the individual can and do produce an effect on the individual's higher nerve-centres, for certainly direct observation shows that these principles, these forces or modes of force alter the facial expression.

Other conditions of the face expressing emotion have been described and illustrated by Darwin. If these expressions be due to muscular action, they must be due to the condition of the nerve-centres; and it is as indicating their conditions that they are of special interest in the pursuit of our special subject. The physiologist and practical physician are principally concerned with the human body as it now exists, and to them it is not a case of the emotion (feeling), causing expression, or the mind showing itself in the face, but rather a condition of the muscles of the face caused by the condition of nerve-centres, and indicating this condition.

Now, passing on to observations of the muscles of the upper extremity. Probably the most expressive muscles are those which move the fingers, i. e. those which produce the finer movements. These finer movements appear more to represent the brain conditions than do the coarser movements, e.g. those of the shoulder or elbow.

It is of course in the free or disengaged hand that we must look for examples illustrating the condition of the brain which governs it. If the muscles be employed in some definite act, such as holding an object, or in an act of manipulation such as sewing, then the movements are directed to accomplish the aim attempted, and are not simply indicative of the condition of the brain, as may be the case with the free hand when unconsciously expressing the mental condition by gesticulation. When, on the contrary, the hands are left free and disengaged, as the hand of the orator which unconsciously expresses by its position or movement the general mental state of the speaker, we have in this muscular movement an expression of the man's mind. It is as reasonable to look for the state of the mind to be expressed in the position and action of the hand engaged in definite voluntary, purposive acts, as to look in the face when the sun is shining full in the eyes, or the lips are engaged in eating, or moved with the other movements of dyspnœa. Still it is true that in either case the manner of performing the act may be indicative of the mental state, but the muscles of the face or hand are not engaged in expressing the mental state.

In Art at the present day we but seldom see the hands represented as disengaged; usually they are painted or sculptured holding some object, or resting on some part of the figure; such are hands engaged or resting from labour, or performing some act of toil, not engaged in expressing the action of the mind.[1]

I have spoken of the finer movements of the hand as the most expressive of the condition of the brain. Movements are spoken of as coarse when performed by large muscles, e.g., movements of the shoulder and hip-joint are coarser than those of the wrist and ankle. Fine movements are performed by smaller muscles, and are confined to small arcs, e.g., slight movements of the fingers; the smaller the arc through which the finger moves, the finer is its movement said to be.

The finer movements of the hand have been spoken of as most expressive of the condition of the brain; some reasons for this statement may now be given. 1. In hemiplegia from

[1] Examples of the disengaged hand are seen in the statues of Cain in the Pitti, Florence; the Venus de' Medici; and the Diana, British Museum.

brain disease the finger movements are most damaged, and the latest to recover. 2. Finger movements are much more damaged by brain disease than by spinal disease.

It may be convenient to commence this study by the consideration of four principal positions of the hand, due to, and representative of the condition of nerve-centres. These may be easily described, and perhaps will be useful in describing conditions of departure therefrom.

In a former paper on "Recurrent Headaches in Children" I described the position of the "nervous hand."[1]

When a very nervous child, or one convalescent from chorea, holds out its hands in front, on a level with the shoulder, and with the fingers spread out, we commonly see this nervous hand. As the palms are turned down, the wrist droops slightly. The metacarpo-phalangeal joints are moderately extended, the first and second internodes being either flexed or kept straight. The thumb is simply extended backwards, and somewhat abducted from the fingers. This position is also maintained when the palms are turned upwards. In such cases there is very commonly finger twitching, of which notice will be taken further on.

With regard to indications of states of the nerve-centres by states of the muscles, there appears to be evidence[2] that in opposite states of the nerve-centres the respective conditions of the muscles are in direct antithesis. Probably also the converse is true, that opposite conditions of the muscles indicate opposite conditions of the nerve-centres. Let us look at the antithesis of the "nervous hand." In applying this principle of antithesis, we must reverse each relative position of the phalanges and joints.

	The Nervous Hand *versus*	The Energetic Hand.
Wrist	flexed.	extended.
Metacarpo-phalangeals . .	extended	flexed.
1st internodes	flexed	extended.
2nd internodes	flexed or ortho-extended	extended.
Thumb, metacarpo-phalangeal	extended	flexed.
„ 1st internode . . .	extended	flexed.
Phalanges relative position .	slight abduction . . .	abducted.

Now we may inquire if this antithesis of the nervous hand be indicative of a condition of the nerve-centres the opposite

[1] 'Brain,' October 1880. [2] See Darwin.

THE NERVOUS HAND.

THE ENERGETIC HAND.

THE HAND AT REST.

THE STRAIGHT HAND.

NOTE.—The hand should be represented as perfectly straight with the forearm.

To face p. 494.

of the "nervous condition." This position is represented in the Diana in the British Museum, and well corresponds with the general forcible attitude of the figure. In one point, however, this hand of the Diana differs from the exact antithesis of the nervous hand, in that the first and second internodes are flexed. The forcible character of this position of a free or disengaged hand is well seen in contrast with the nervous hand of the adjacent Venus; and it was in comparing these that I saw the one to be the antithesis of the other.

This energetic or powerful hand is probably in life seen only under certain mental conditions, or as it is preferable to say in certain conditions of the brain producing what we call mental states, such states of the mind giving energy and the feeling of strength. I do not know that it is to be seen as the result of any pathological condition of the brain. It is, I think, seen in forcible expressions of feeling; and in astonishment, as expressing which it is figured by Darwin.

The most natural position of the hand is doubtless that of rest, and here analysis shows all the joints in the position of flexion. The hand may be seen in perfect rest during sleep, or when the man is resting, engaged in quiet unexciting conversation. The following is the analysis :—

The Hand in rest		*versus* Hand in fright.
Wrist	flexed	extended.
Metacarpo-phalangeals	flexed	ortho-extended.
1st internodes	flexed	ortho-extended.
2nd internodes	flexed	ortho-extended.
Thumb, metacarpo-phalangeal	flexed	ortho-extended.
„ 1st internode	flexed	extended.
Phalanges, relative position	abducted	abducted.

This analysis represents a hand in complete flexion and its antithesis a hand in general extension or ortho-extension. The term ortho-extension is used to imply that the joint is so far extended as to place both the bones constituting the joint in the same straight line. If extension be full or complete, the bones form an obtuse angle with one another.

(To be continued.)

TETANUS.

BY SURGEON-MAJOR J. J. L. RATTON, M.D., MADRAS.

In a paper published in 'Brain,' Part VIII., January 1879, I endeavoured to show that peripheral nerve-irritation in some part of the body is invariably a factor in the causation of tetanus. That there are other factors at work, predisposing and exciting, "goes without saying," and to these points I will recur; but for the present I confine myself to peripheral nerve-irritation, believing that the subject requires further elucidation. There can be little doubt that the nerves implicated in tetanus are the ordinary motor or mixed nerves. This seems to have been proved, clinically and experimentally. The important question now arises, what is the nature of the irritation? Is it mechanical or tactile; pathological or painful; or a combination of both? Our limited knowledge of nerve-physiology and pathology would lead us to the conclusion that it must be either one or the other, or a mixture of the two. On the threshold of this investigation we are met by the difficulty of determining how external impressions are conveyed by the nerves. We know that the nerves are conductors. We know that they consist essentially of a semi-fluid conducting medium, contained in tubes, and strengthened by fibrous sheaths. We also know that it is not essential to the performance of a reflex act that the nerve implicated should conduct to the brain; but we do not know how, or what, the nerve conducts. We do not know whether nerve-force is one of the natural forces known to science, working through living cells, and thereby altered in its reactions; or whether it is a new and unknown quantity. We never shall know, probably, how a man lifts at will his little finger, without antecedent cause of any kind, except his own volition.

But this is a higher flight than necessary here. The question is, how are tactile sensations transmitted to the nerve-centres through the centripetal nerves?

I incline to the belief that tactile impressions are a form of motion; that as light and sound are appreciated by the eye, and ear, and brain, by their vibrations, so the appreciation of external objects, by the sense of touch, is a question of vibrations. And to this I have been led not only by analogy, but also by a very simple experiment, which illustrates the matter in a coarse exaggerated way. Any one who draws his finger over a rough surface, and then over a smooth one, notices at once the difference of vibrations communicated to the finger. It is by these vibrations he appreciates the one as rough, the other as smooth. And, *vice versâ*, if a person touch but lightly different objects, with his eyes blindfolded, he will be unable to give their most prominent physical characteristics until he has, by some little friction with the fingers, produced the corresponding vibrations. There is nothing very new in the proposition that vibrations, mechanically produced, by feeling with the fingers or by surface-contact with external bodies, are propagated, unchanged perhaps, through the diffluent centres of the nerve-tubes, to the nerve-centres, or to the brain, where they are appreciated and expended in the convolutions.

As a matter of fact, the waves of sound, with which we may compare the vibrations of objective feeling—though the latter are probably larger and more violent—travel at the rate of 4000 feet per second in water, and in solids with varying velocities, up to 16,000 feet per second. If we suppose tactile vibration transmitted at the lower rate, 4000 feet per second, through the semi-fluid matter of the nerves, a person six feet high would have cerebral consciousness of an object touching his foot in the 1–666th part of a second, quite fast enough to meet the facts of the case—the requirements of the hypothesis.

That the brain is partly formed to appreciate vibrations is probable from its convoluted shape, and is proved by the perception of light and sound. Here we have to deal with molecular vibrations, or waves, so delicate and ethereal, that special

organs, the eye and ear, have been developed for their reception. There can be little doubt that the waves of sound, caught upon the tympanum, are passed on to the brain, diminished in intensity it may be, but not otherwise changed. The science of acoustics is a monument of the appreciation of vibrations by ear, and eye, and brain. Heat and cold are also modes of motion. Moreover, heat, light, sound, and ordinary mechanical motion—I might even include electricity—are interchangeable modes of motion, and convertible one into another. I suppose that the molecular vibrations of heat and cold, different from others in wave-length, or rapidity, are conveyed to the brain by the nerves, acting as simple conductors, in the same way that ordinary tactile sensations are conveyed.

All this leads to the conclusion that man derives all his impression of external objects by vibrations, and that his nerves are, in one sense, merely conductors of vibrations, just as telegraph wires are conductors of electricity; though they have other, higher, and vital functions as well. It may be well to repeat here that these views are held only with reference to the centripetal nerves. How these vibrations react upon the nerve-centres, and what becomes of the nerves, and centres, when the individual perpetually bombarded by vibrations is asleep, I confess I do not know. This is a matter closely linked with the great question of how the will acts, with which I have no wish to meddle.

Before proceeding to apply the above hypothesis to the solution of the problem as to how peripheral nerve-irritation produces tetanus, let us see what pain is; for, as before stated, the irritation may be either tactile or painful, or both combined.

Pain, there is reason to believe, is an affection of the vaso-motor system of nerves. C. D. Radcliffe, M.D.,[1] looks upon pain as being the result of *irritation* of the vaso-motor nerves and contraction of the capillaries. He points out that in all diseases when inflammation, denoting *paralysis* of the vaso-motor nerves, begins, pain diminishes, except where fibrous coverings prevent swelling and cause tension. He

[1] Reynolds' 'System of Medicine,' vol. ii.

instances the pains in the cold stage of fever, and at the beginning of an attack of gout, before heat and swelling are developed, and other cases.

On the other hand, most men connect pain with inflammation and paralysis of the vaso-motor system. Heat, redness, pain, and swelling are the most prominent symptoms of inflammation; and it is hard to say in what order these symptoms appear, so closely do they occur together.

Lateral section of the spinal cord causes motor paralysis, with increased temperature and sensibility, on the side cut; loss of sensation with diminished temperature and no paralysis, on the opposite side. It would seem from this, Dr. Radcliffe remarks, that the injury had acted upon the vaso-motor nerves contained in the cord, as well as upon the common motor and sensory nerves, causing paralysis of the vasor-motor nerves on the side on which there is increased temperature and sensibility, and irritation of vaso-motor nerves on the side on which there is diminished temperature and anæsthesia. The value of this experiment depends upon the mode of distribution of the vaso-motor nerves in the cord. As they accompany the circulatory system, it is possible that they do not decussate, in which case the significance of the experiment would be completely altered. Be this as it may, pain has long been recognised as a disturbance of some kind of the vaso-motor or circulatory system, and even in its purely nervous forms, as the various neuralgias, excepting always hysteria, it is probably a form of inflammation.

Dr. S. Weir Mitchell, of Philadelphia ('BRAIN,' Part III.), chilled a limited portion of a mixed nerve until touch was lost; at this point the least tap on the nerve caused motion of all the muscles supplied, and some pain, showing that pain-sensation passed the frozen part, whilst tactile sensibility could not; they are therefore probably different things. His close study of therapeutic neurotomy in several cases leads him to ask, "but why should this operation, which annihilates touch, leave pain-power unaltered in the regions under the skin?" I infer it is because pain is an affection of the vaso-motor nerves, which have extended connections through the circulatory system.

It is worthy of remark here that the sensation of pins and needles, so common in the hands and feet, after prolonged pressure on the axillary and sciatic nerves, is due to arrest of blood-circulation and paralysis of the vaso-motor nerves.

If there be any truth in the supposition that pain is connected with disturbance of the capillary circulation, and not with peripheral irritation of the cerebro-spinal axis, we should expect to find it absent, or at least playing a very subordinate part in the causation of tetanus. It is a remarkable fact that pain and inflammation are generally absent from the wound which causes tetanus. Often, indeed, tetanus shows itself when the wound is kindly healing, or has healed. This fact has attracted the attention of most authors on this subject, and John Hunter, amongst the rest, remarks, that the irritability of the nervous system which developes into tetanus is not due to the presence of inflammation, since it appears only when the latter has been removed. It would appear from the records of tetanus that there is no connection between the frequency of tetanus cases and the painfulness of wounds. Neuralgias affecting, most painfully, the peripheral extremities of various nerves, never end in tetanus. The same thing may be said of gout. Neuromata, or subcutaneous tumours, are not known to cause tetanus; yet these are amongst the most painful affections to which man is subject.

I believe then, in answer to the question proposed at the head of this paper, that we may leave pain aside, at least for the present, and confine ourselves to the study of tactile irritation to elucidate the etiology of tetanus; not that I would exclude pain entirely, but because I think that it plays a subordinate part.

Before leaving the subject of neuralgia, it may be noticed that the late F. E. Ainstie, M.D.,[1] instances a number of cases of neuralgia resulting from injuries of various kinds to mixed nerves; even foreign bodies, as a bit of glass, have been entangled in the nerve, and caused neuralgia for months, but never tetanus. He makes the following comment: "So far as I know, it is only when a nerve-trunk of some size is injured that neuralgia is a probable result. Wounds of the small

[1] Reynolds' 'System of Medicine,' vol. ii. art. "Neuralgia."

nervous branches of the fingers, for instance, are very seldom followed by neuralgia. I have no statistics to guide me as to the effect of long-continued *irritation* applied to one of these small peripheral branches, but it is probable that that might be more capable of inducing neuralgia. As far as my own experience goes, however, it would appear that a more common result is convulsion of some kind, from reflex irritation of the cord."

It is observable that the nearer we get to the peripheral extremities of the nerves, the more markedly reflex is the effect of irritation. A cold plunge that sets a man shivering and shaking, and his teeth chattering—mark the tetanic symptoms—affects only the peripheral extremities of the cutaneous nerves.

JAS. J. L. RATTON, M.D.

METHODS OF PREPARING, DEMONSTRATING, AND EXAMINING CEREBRAL STRUCTURE IN HEALTH AND DISEASE.

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II.

Physical Properties of Grey and White Matter.

In the introductory chapter to his great work on Pathological Anatomy, Rokitansky has said:—"Just as there is a general and a special anatomy, physiology, pathology, so there must in like manner be a general and a special pathological anatomy. The former treats of anomalies of organisation, the latter of the special anomalies of individual textures and organs." It appears to me that in the examination of the diseased brain this natural classification must be kept in view. The student should be taught to appreciate the various general anomalies of organisation as they present themselves in the brain; and the methods to be adopted in estimating the degree, extent, and significance of such physical deviations. It is not, of course, within our scheme to detail the various diseases of texture to which the brain is subject, but rather to place the student in possession of those indications of healthy and diseased tissues which are presented by alterations in the physical properties of grey and white matter. The elements of a physical diagnosis, so to speak, are placed at his disposal, and hints thrown out for his guidance, where it is thought a wrong construction might be attached to the physical signs.

It will be found, however, convenient to illustrate the more profound anomalies in consistence, colour, specific weight, and other physical qualities by reference to some of the special diseases of texture to which the nervous centres are liable, and

[1] 'Pathological Anatomy,' Sydenham Soc., vol. i.

which may serve as typical cases wherein the student may acquire a practical acquaintance with the methods of physical research.

The fundamental physical properties of the nervous tissues with which it is necessary we should acquaint ourselves are briefly as follows:—Consistence, colour, volume and weight, both absolute and specific. Each in turn will now claim our close attention.

A. *Consistence.*—The firmness, solidity, or consistence of a texture depends upon the cohesive force exerted by the individual elements of its mass, and to which are due the varying degrees of toughness, hardness, and resistance on the one hand, or of softening friability and liquefaction on the other hand, to which they are liable. The consistence of a compound tissue so complex as the brain is necessarily subject to very great variations, since its individual constituents vary amongst themselves greatly in their physical properties. An organ composed of nerve-tubuli, nerve-cells, of a connective framework and an elaborate system of arteries and veins, the whole pervaded by nutrient fluid, must be subjected to great modifications in consistence due to alterations in the relative proportions of one or other of the constituents. Hence it is that we get such variety in the structural firmness of brain-tissue, not only in different animals and in the brain of man, but in different regions of the same brain. It is this textural difference betwixt grey and white matter which explains their varying degrees of consistence, and the same explanation pertains to the firmness of the occipital as contrasted with the frontal lobe; of the central grey ganglia as contrasted with the grey envelope or cortex of the cerebrum. The grey cortex, consisting of a vast assemblage of nerve-cells and their protoplasmic extensions, imbedded in a most delicate web of connective tissue, with a complicated vascular apparatus, has a far lower degree of consistence than the white matter which owes its solidity to large medullated nerve-fibres and an abundant and coarser connective matrix. A moment's consideration will suffice to indicate to the student that the forces which in disease modify textural cohesion must come from *without* or from *within* the structural elements; in other words, the tissue

elements may be forced asunder by fluids pervading the texture and by the intrusion of adventitious bodies, or by nutritive changes in the elements themselves, whereby a quantitative or qualitative transformation may be induced. *What conditions, then, modify the normal consistence?*

1. The forcible infiltration of nervous texture by serous fluid, by plastic exudates, or by blood, will in the first place tend to destroy all textural cohesion, and consequently produce reductions in consistence; whilst further changes in the effused blastemata due to organisation will result in increased firmness or induration.

2. Chemical changes in the constitution of the individual elements, according to their nature, may tend to produce increase or reduction of consistence, e.g., fatty, amyloid, and calcareous degenerations, the nutritive anomalies due to inflammation, decomposition of the structural elements due to putrefactive changes.

3. Apart from the above qualitative anomalies of nutrition, we must not overlook the quantitative element which sometimes exists alone or in combination with the former. Such are the forms of hypertrophy and atrophy due to an increase in size and number of constituent elements on the one side, and on the other, in the decrease in size, number, and diminution in the number of the tissue elements.

The student must guard against the fallacy of regarding density and consistence of a tissue as in any way mutually convertible terms, for although in a large number of cases increased density may co-exist with increased consistence of the brain, conditions occur where with increase of density there is really a diminution in consistence. Thus, in inflammatory conditions of the brain-tissue where plastic exudates have forcibly reduced the cohesion of the mass, the specific weight of the part is notably increased. Hence the specific gravity of cerebral tissue will afford us no exact and reliable guide as to degrees of consistence; and, in fact, we have no more exact gauge of consistence of texture than the rough-and-ready methods afforded by the sense of sight and touch. How do we judge of the relative cohesion of texture?

a. By the eye we note deviations from the natural compactness of an organ, or the maintenance of its normal contour. Contrast the brain of a senile dement or of a general paralytic with the compact firm brain of an epileptic; or the same organ in warm weather and in a state of incipient decomposition with the recent brain removed from the skull in cold frosty weather.

b. By the sense of touch we note relative resistance to pressure and the compressible and lacerable character of the tissues. Especially note this with respect to the central commissural tracts, basal ganglia, fornix and septum lucidum.

c. By its resistance to a graduated force, such as the rough gauge afforded by a stream of water falling from a variable height (applicable only to cases of much reduced consistence). Any case of white or inflammatory softening may thus be tested.

d. By its resistance to section (applicable to the slighter degrees of reduction and all degrees of increased consistence). Contrast the resistance to section presented by the medulla, pons, cerebellum, cerebral hemispheres, and ganglia at base.

Observe that we gain by these means not only a satisfactory (although approximate) gauge of consistence, but that the methods vary in their relative values in different cases. The student should take every opportunity of rendering himself perfectly familiar with the general consistence of healthy brain prior to post-mortem change. Let him remove a normal brain for careful study, and note the following points:—

The Normal Brain.—Observe that upon removal from the cranial cavity it preserves its normal contour, although it has lost its natural support—the cranial bones. It is plump, rounded and compact, giving a general impression of firmness and solidity, ere we gauge its consistence further by handling. The hemispheres are closely approximated by means of the strong commissural band—the corpus callosum, showing no tendency to fall apart from rupture of the commissure, such as is so frequently seen in the degenerated and diseased brain.

Upon separating the cerebral hemispheres with the hand the corpus callosum appears intact, and offers fair resistance to the blade when dividing the hemispheres along this course. The individual lobes maintain their characteristic forms, their salient margins and relative positions; each convolution remains firm, plump, and in close contact with its neighbour, whilst the arachnoid investment gives a uniform, smooth, glossy aspect to the consolidated organ beneath. A section across the centrum ovale reveals a similar condition—observe the general solidity of the parts; how perfectly relative positions are maintained; the absence of any gaping of the sulci; observe also that the white substance cuts with a clean section, and shows no tendency to cling to the blade. Next open up the lateral ventricles, and observe the rounded firm aspect of the intraventricular portion of the corpus striatum and thalamus. Examine the fornix and septum lucidum—parts especially prone to softening in certain affections of the brain. Pay special attention to the relative consistence and size of the corpora quadrigemina, the cerebral peduncles, the pons and medulla. Now we must be prepared to meet with all varieties of consistence betwixt the above-described normal firmness of health and extreme alterations produced as the result of disease. The consistence, as we shall illustrate further on, may be so far reduced that the cerebral substance may be perfectly diffluent, and may be poured away like thin cream; or, on the other hand, it may possess the firmness, aspect and character of the hard-boiled white of egg; or even cause the knife to creak in cutting through patches of almost fibro-cartilaginous induration.

Reductions in Consistence.—Let us now consider briefly the appearance and significance of those abnormal conditions of cerebral tissue which lead to softening, dwelling only upon those which are most likely to present themselves frequently to the student's examination. The conditions best illustrative of reduced consistence are the following:—

1. Putrefactive changes.
2. Softening as the result of disease.
   a. White softening.
   b. Yellow or gelatinous softening.
   c. Red or inflammatory softening.

3. Simple œdematous conditions.

1. *Putrefactive Changes.*—Now these changes in the brain occur sooner or later after death, according to the temperature and humidity of the surrounding atmosphere, modified, of course, by the morbid conditions of the cerebral texture and contained fluid. It is on this account that the report of an examination of the brain should invariably be accompanied by a statement of the number of hours after death at which the examination was made, the average temperature of the post-mortem room, and the condition of the atmosphere as to humidity. Fremy, in an elaborate research in 1841 ('Ann. Chim.' 2, 463), arrived at the conclusion that the ordinary form of softening of the brain was analogous in its production to the putrefactive process occurring post-mortem; an opinion which has not been supported by later researches into the chemistry of the brain. The student should learn to discriminate between the softening due to this cause and that due to morbid alteration of texture. He will be guided here by observing that the softening is general throughout the encephalon, and attended by much blood-staining by the evolution of offensive gas, and the presence of the latter within the blood-vessels and beneath the membranes. He must also take into account the absence of any morbid condition tending to produce softening, and the conditions, atmospheric and otherwise, to which the brain has been exposed since death. It is essential also to keep in mind the fact that various diseases predispose to early putrefactive changes whilst the mode of death may expedite or defer the same.

How are these putrefactive changes to be avoided?

To retard putrefactive change when the brain cannot be immediately examined, it should be placed in a cold room with a damp cloth thrown over the hemisphere to prevent desiccation of the outer cortical layers; or better still, it may be enclosed within an ice safe. The student can readily extemporise for his own use such a safe, by placing the brain in a jar or box which fits into a larger box, leaving a space of an inch and a half around between the outer and inner box, into which ice is packed. The outer vessel must be thickly and completely enclosed in felt. Such a contrivance he will find

of great service in the summer months, when it is difficult to keep anatomical subjects fresh for many hours together. Portions of brain reserved for microscopic examination should be transferred immediately to methylated spirits or Müller's fluid, or better still, to methylated spirits coloured of a dark sherry tint by tincture of iodine. For the fresh methods of examination should any delay occur, we must trust to the ice safe for the preservation of our material—preservative fluids being scarcely admissible. As regards the latter, however, the strong solution of acetate of ammonium recommended by Sankey[1] is perhaps the best which can be used (sp. gr. 1·040).

Indications of Softening.—A general reduction in the consistence of the brain will usually reveal itself at first sight to a mere superficial glance. The whole brain assumes a flattened squat appearance, and the hemispheres diverge. The student will find the former due to extension of softening to the central portions of the hemispheres which support in the normal state the convolutionary surface folded over them, whilst he observes the commissural band connecting the hemispheres (corpus callosum) tends to split asunder in a longitudinal direction, i.e. across the course of the great bulk of its softened fibres. Proceeding to handle the brain he finds the convolutions flabby to the touch and presenting less resistance to pressure—the whole brain is less able to resist the ordinary force of gravity, and is therefore strongly contrasted with the plump, erect, and compact aspect of the healthy brain. Let the student examine the brain of an advanced general paralytic and he will find this condition well represented. Cut across the hemisphere of such a brain so as to expose the *centrum ovale*, and it will be observed that the brain-tissue clings to the blade with unusual tenacity unless the latter be kept constantly wet by water or spirit. As a result the cerebral tissues both grey and white are lacerated and tear away in shreds, leaving an unmistakable softened rottened aspect of the surface of the section. This tearing away of brain-tissue is apt to give one a false idea of the *comparative*

[1] 'New Process for Examining Brain Structure,' West Riding Asylum Reports, vol. v. p. 192.

coarseness of texture; thus in the brain of a general paralytic, the adherent, thickened membrane and softened cortex cannot be cut without leaving a coarse irregular surface, unless the precaution be taken of using a very sharp blade and keeping its surface constantly wet. Let us now examine more carefully the special form of softening, known as white softening.

2. *White Softening of the Brain.*—We may meet with this condition as one generally diffused through a hemisphere or in patches limited to a convolution, the area of softening being often no larger than a pea, although more extensive tracts are usually involved. In the first place, suppose we have before us a brain in which the greater part has thus suffered. The affected hemisphere presents to the touch a soft, boggy feel, sometimes communicating an almost tremulous, fluctuating sensation to the finger if the disintegration be extreme. The brain will be with difficulty removed, the disintegrated tissue tending to burst through the softened walls which confine it, and more especially is this the case in the neighbourhood of the anterior perforated space and commencement of the Sylvian fissure. All unnecessary manipulation of such a brain should be avoided, as it is attended with danger to the textural continuity of internal parts, which should also be examined at as early a stage as possible. Observe the lateral divergence of the hemispheres and their flattened aspect above. By gently drawing the hemispheres apart and introducing the blade of a knife into the longitudinal fissure a section may be made outwards across the affected hemisphere, and thus removing the vault we find the centrum semi-ovale occupied by a mass of broken-down medulla—all normal cohesion being destroyed by the presence of a large quantity of interstitial serous effusion. The disintegrated substance will probably have the appearance of soft watery or creamy pap readily raised upon the blade, or it may be absolutely diffluent, flowing away as soon as the resistance offered by the confining grey cortex is overcome. It may, however, retain a certain amount of cohesion, yet break readily and wash away on placing it beneath a stream of water. The ganglia at the base may be implicated, and the septum pellucidum, grey commissure, fornix and corpus

callosum will generally be found softened or even broken down, the ventricle containing more or less serous fluid. Now such a brain as we have just been considering will be met with in acute hydrocephalus, and in thrombosis or embolic plugging of the main cerebral arteries. With such a case before him, the student should pay attention to the following points:—

The degree of softening.—Does it resist a stream of water?
Does it render a stream of water milky or break down readily under it?
Is it from the first diffluent?

Extent of area involved.

Œdematous condition of the parts involved.

Turbidity or clearness and colour of effused fluid in the ventricles.

In noting the degree of softening, learn to appreciate how very short a period is requisite after arrest of blood supply, for the production of extensive nutritive changes. With what object do we endeavour accurately to map out the *extent* of the destroying lesion? Our object is to detect that portion of the vascular apparatus involved if the softening be due, as it most frequently is, to plugging of a large branch. This the more frequent source of extensive white softening requires therefore for its full comprehension an acquaintance with the vascular areas of the brain.

On noting the appearance of the serous effusion in the ventricles, so often accompanying white softening, the student will be guided towards concluding as to the presence or not of inflammatory action in the meninges or in the substance of the brain itself. Thus if the fluid be more or less turbid and discoloured (as the result of a plastic exudate and macerated cerebral substance), we may reasonably suspect an inflammatory condition, and expect to find upon microscopic examination exudation corpuscles, compound granule masses and other products of inflammation. Such are the conditions found in acute hydrocephalus, distinguishing it from a purely non-inflammatory form of hydrocephalus, which occurs where the fluid, though perhaps slightly turbid from broken-down cerebral tissue and shreds from the macerated lining membrane

of the ventricles, presents no inflammatory material amongst the débris.

Limited Foci of Softening.—Acquaintance with the peculiar arrangement of the cerebral blood-vessels soon leads the student to infer that very minute tracts of softening may result as the effect of thrombosis or embolism, the area involved being dependent upon the size of the clot and the site of its arrest or formation. It is necessary that the mechanism of white softening of cerebral tissue, as a result of thrombosis or embolism, be thoroughly understood. The process may be best elucidated by the diagram below.

DIAGRAM ILLUSTRATIVE OF THE EFFECTS OF EMBOLIC PLUGGING (AFTER RINDFLEISCH).

a a. Portion deprived of its blood supply by the Embolus E. A. Artery. V. Vein filled with blood-clot. The arrows indicate the collateral channels which lead to a hyperæmic zone around the occluded vessels.

Beyond the obstructed artery is the wedge-shaped area of its distribution, now anæmic and consequently deprived of its functional power. Below the embolus are seen swollen branches, which tend to establish a collateral circulation. If this fails, we get as a result engorgement of the latter vessels, and a congestive vascular zone surrounding the central mass. The tissue here becomes swollen and œdematous and minute hæmorrhages are apt to occur, whilst the whole central and peripheral texture becomes broken up by the effusion, and a true necrosis occurs of the tissue forming the area of distribution of the nutrient branch which has been plugged.

All inflammatory foci have as a result the production of a similar congestion and œdema of the surrounding texture,

accompanied by white softening; and as new growths or tumours are frequently the site of such inflammatory states, we may find them imbedded in a cavity containing broken-down tissue.

In addition, therefore, to the appearances which the student has been directed to look for in white softening of the brain, let him also note—

a. Any obstruction to the vascular supply by thrombi, emboli, or adventitious products pressing upon the vessels from without.[1]

b. The establishment of a collateral circulation.

c. The presence of inflammatory foci, to which the white softening is secondary.

Yellow Softening.—In the course of his pathological studies the student will frequently meet with appearances which, whilst significant of cerebral softening, differ much from that just described. He will find frequently in the white substance of the hemispheres, less frequently in the cerebellum, and rarely on the convolutionary surface of the cerebrum, a focus of softened tissue, varying in size, but scarcely ever larger than a hen's egg, and characterised by its bright straw-yellow colour and soft gelatinous consistence. Pressure causes a yellowish fluid to exude, and when cut across it is found to be somewhat sharply defined from the pale swollen and œdematous tissue around. The student will recognise in these softened areas the lesion termed yellow softening of the brain.

Pushing on his examination, he will discover that these spots may be primary, or, on the other hand, secondarily induced around adventitious products, such as tubercle, cancer, cysts, hæmorrhages, or around a patch of inflamed tissue. If secondary to adventitious growths, there is, as Rokitansky points out, an intermediate zone of red softening betwixt the growth and the yellow softened exterior.

It is important that these patches should not mislead the student. He must from the outset regard them as identical in nature with the first form of softening described, the difference in colour being due, according to some, to altered blood-pigment, but according to Rokitansky, to a peculiar chemico-pathological process.

[1] Plastic exudates, tubercle, syphilitic gummata.

The following facts are to be noted:—

a. The fluid is usually intensely acid.

b. There is little or no vascularity surrounding the patch.

c. Every degree of tint may be found, from white softening up to the bright yellow of typical yellow softening.

d. Colour differs from the rusty or ochre-yellow tint seen in old apoplectic cavities, &c.

e. Microscopic examination fails to reveal inflammatory products.

Red or Inflammatory Softening.—My reader will now be prepared to infer that profound alterations in consistence may attend inflammation of the nervous structures, and he will find little difficulty in recognising foci of inflammatory softening. He will occasionally meet with portions of acutely inflamed tissue in the medullary strands of the cerebrum, varying in size from a hazel-nut to that of an orange, or still larger; yet by far the more frequent site of such inflammatory foci will be the grey cortex of the cerebrum and the basal ganglia. Examine a brain which presents a focus of inflammatory softening in the medulla of one of its hemispheres. Note the following facts:—The *softening of texture* is accompanied by modifications of colour and general moisture. Thus the increase in vascularity gives the affected part a streaky red aspect, often profusely besprinkled by puncta vasculosa; the œdematous infiltration of texture is recognised in its swollen and moist aspect. Where the engorgement has reached a high degree, note the numerous minute extravasations of blood, the streaked or punctuated capillary hæmorrhages.

Now the student must be fully impressed with the fact that all the above characters may be developed in the course of extreme congestion. What impresses upon the affected part the stamp of inflammatory action is the presence of exudates, which modify the above described appearances in two directions:—

a. The dark red becomes of a paler and more uniform hue, whilst exudations of lymph still further modify the aspect.

b. Alterations in consistence occur; rapid softening or solutions of textural continuity varying with the plasticity of the exudates.

Observe, therefore, that the essential features of red inflammatory softening are profound alterations of consistence, associated with the pouring out of inflammatory exudations.

In advanced stages of red inflammatory softening, the student will, therefore, find the tissues broken down into a reddish pulp, or having a brownish or rusty aspect, washing away freely when held beneath a stream of water. He will also note the results of swelling from congestion and œdema, viz., the flattening of the convolutions of the hemispheres from pressure against the internal table of the skull, and a well-marked prominence of the surface of a section of the inflamed and œdematous textures.

Examine a patch of inflammatory softening in the cortex, and note :—

Inflammatory foci situated in the cortex will implicate by contiguity the superimposed membranes. Observe in these cases that the grey matter is more deeply coloured, and is still more swollen, moist, and softened than when the white matter is involved. The far greater vascularity and the naturally looser texture of the grey matter account for this difference. Next direct attention to the state of the membranes. The pia mater cannot be removed without peeling off with it a layer of the softened cortex, whilst the meshes of the membrane are found infiltrated with inflammatory products and are both thickened and œdematous. As regards these apparent adhesions of pia mater, observe that the brain substance may tear away with the removal of the pia mater from two causes :—

1st. Mere unnatural softening and loss of textural cohesion of the grey matter, without any true adhesion to the membranes as in early inflammatory and congestive stages, &c.

2nd. Actual adhesion, inflammatory in origin, may be the cause of this tearing of the cortex.

How are we to distinguish the lacerable cortex in the former case from the genuine inflammatory adhesions of the membranes? In the former case the surface left by the irregular shreds torn off is soft, pappy, and uniformly smooth or homogeneous; in the latter case the surface is studded with numerous perforations—the apertures of perivascular

channels from which the blood-vessels have been withdrawn. These channels will be seen much dilated from the prior engorgement of the blood-vessels, and the surface generally has a peculiar worm-eaten appearance.

Fallacy to be avoided.—The student must not regard all cases of encephalitis as necessarily attended by injection and discoloration of tissue. The larger number of cases, in fact, show little or no discoloration; and even where softening has advanced to an extreme degree, the naked eye fails to appreciate in the uniformly dull, white pulpy material its inflammatory origin.

What criterion have we here for determining the nature of the lesion? Recourse must be had to the microscope and specific gravity bulbs.

The *microscope* shows us nuclei, nucleoli, pigment, and compound granule cells amongst broken-down cerebral tissue infiltrated with leucocytes and effused lymph along the course of the blood-vessels. The specific gravity test (to be hereafter described) will indicate an invariable increase in specific weight where inflammatory exudation has occurred. On the other hand, non-inflammatory white softening shows an invariable reduction in specific gravity.

By the freezing methods it will be possible to obtain fair sections through inflammatory patches of greatly reduced consistence, and much valuable information will be thus obtained; but in all cases of extreme softening all that can be hoped for is obtained by raising a little of the creamy pulp upon the scalpel, and transferring it to a slide and using a thin glass cover. Sections through the membranes and cortex, in cases where these are involved, will prove highly instructive, and should never be neglected. They should be studied in the fresh state, and the student become thoroughly familiarised with the morbid appearances ere he attempts to mount stained sections of such lesions for permanent preservation. Coarse blood-vessels, coursing through the inflamed patch should be drawn out and examined microscopically, noting abnormal conditions of their coats and sheath.

(*To be continued.*)

Critical Digests and Notices of Books.

Maladies de la Moëlle. Par A. VULPIAN, Doyen de la Faculté de Médecine, Membre de l'Institut, etc. etc. Grand 8°. Paris: Octave Doin; pp. 512.

THE volume before us contains a series of lectures on some of the diseases of the spinal cord, delivered by the author in his capacity of professor of experimental pathology. M. Vulpian's great authority, both as a physiological and clinical observer, as well as his felicity of expression, make the reading of this book a duty as well as a pleasure to all those interested in spinal pathology. We regret, however, that he should have preserved the form of lectures in the present work. The subject-matter is wofully cut up; and yet considerable changes must have been made in the original 'Leçons.' For instance, Lecture XIII. occupies twenty pages, devoted to the incipient stage of locomotor ataxy; whereas in Lecture XIV. the first stage is concluded, and the second and third discussed and illustrated with cases, the whole occupying two hundred pages!

Experimental pathology, though bound within very narrow limits, and unable to reproduce in animals most of the diseases observed in man, has thrown floods of light upon certain obscure symptoms of spinal lesions. But it has done more than that: it has given origin to a stricter spirit of clinical observation and educated the mind in the framing of scientific hypotheses, and so rendered possible the solution of problems to which its methods were not directly applicable. Indeed it is doubtful whether the cultivation of this mental discipline is not of greater value than the actual establishment of facts; and it is to be regretted that the defence of vivisection against the

blind fury of the sentimental fanatics who revile it and its devotees, should not have been more generally based upon this higher ground, rather than its legitimacy vindicated by an appeal to the individual discoveries to which it has led. Though the leading thought of Prof. Vulpian's work is the explanation of the various symptoms observed in the diseased human cord through the data of experimental inquiry, he has given a very large share to the purely clinical aspect of the question; and though the various diseases touched upon in the present volume are not all as fully discussed and illustrated with typical cases as locomotor ataxy, for instance (which occupies about half the book), and the work has no claim to be a systematic exposition of spinal complaints—yet theory is so constantly verified by references to practice, that no one will arise from the perusal of these pages without being a better physician as well as a wiser pathologist.

The first subject discussed is compression of the spinal cord, sudden or gradual. The symptoms in both cases involve, eventually at least, similar consequences; it may be observed, however, that in acute compression sensory troubles are usually not conspicuous. Chronic compression occurs as a consequence of a variety of pathological states, such as growth of various kinds, thickening of the dura mater, effusions into the canal, etc. Caries of the vertebræ, or Pott's disease, offers a good example of it. The actual cause of the compression here has often been stated to be the deformation of the column; but this is certainly erroneous in the great majority of cases, for on the one hand, compression is sometimes present where no deformation is noticeable; on the other, considerable deformation may occur without any signs of compression being developed. The compression is due to an external pachymeningitis, as shown by Michaud. The external layers of the dura mater are inflamed by contiguity, and become the seat of a fibro-nucleated proliferation, and a caseous layer usually results from the retrogressive changes in the neoplasia. This appears to the naked eye as if merely superimposed upon the internal layer of the dura mater, but the microscope easily displays its true nature. Prof. Vulpian does not agree with those who refer all the pain often associated with this pachy-

meningitis to pressure upon the posterior roots. He appeals to the results of his experiments, which have shown the dura mater itself to be acutely sensitive, especially when inflamed. Of course it would be unsafe to base this opinion upon mere manifestations of suffering on the part of the animal experimented upon. But we have two ways of proving the irritation of sensory nerves; the one, by the increased vascular tension with heart-beats diminished in number, but increased in strength; the other, by the dilatation of the pupil. By both these methods we can prove the dura mater to be sensitive.

The acuteness of pain in Pott's disease may induce a pseudo-paraplegia, which, however, with advancing pressure, is soon converted into a true paraplegia. This interference with the transmission of voluntary impulses through the cord is often entirely dissociated from any corresponding anæsthesia—an important diagnostic feature in cases of compression. To explain this fact by the assumption that pressure is mainly localised on the anterior aspect of the cord is erroneous. It is much rather due to the fact that the conductivity of the grey matter, alone necessary for the preservation of sensation, is less readily destroyed than that of the white fibre columns. There are, however, many cases where hypæsthesiæ and paræsthesiæ of varied character testify to some alteration of the grey matter.

The increase in the reflex activities of the lower segment of the cord, when cut off from the higher centres, has been attributed by Setschenow to the suspension of the inhibiting influence of a special centre, which he placed in the isthmus. But, as Vulpian shows, direct stimulation of the isthmus does not suspend the spinal reflexes; and their exaggeration increases with the distance from the brain at the point at which the cord is cut. With reference to the augmentation of reflexes, amounting to tonic rigidity, observed in the later stages of compressive paraplegia, we note that it is frequently preceded by a period of muscular resolution. Possibly, descending sclerosis plays a part in its causation; but we may hope that a cure is not impossible even when rigidity is well marked.

According to the locality and nature of the lesion (irritative or paralysing) the vasomotor and trophic symptoms (as well as

those connected with micturition and defecation) will vary. Of special interest is the compression of the cervical enlargement, which brings about brachial paraplegia. In a frog this can be easily illustrated by inserting a small piece of wood along the spinal canal. The animal then swims easily with its legs, while the arms hang helplessly down. Generally speaking, every compression acts most markedly upon the nerves originating at the point compressed, the explanation of this fact being possibly that its influence is more readily exerted upon those motor fibres emerging from the grey matter to form the anterior roots, than upon those constituting the lateral bundles from the cerebral centres.

A paradoxical result often observed is that compression of the mid-dorsal cord frequently involves paresis of the arms (recurrent paralysis). In connection with this point it must be remembered that the vasomotors of the upper extremity arise from as low as the level of the seventh dorsal pair. There is always some degree of myelitis in Pott's disease, and this may give rise to more or less secondary damage in the cord, ascending or descending.

Compression of one-half of the cord gives rise to the well-known symptoms described by Brown-Séquard. It must be remarked that in spinal, as well as cerebral hemiplegia, the arm is always more involved than the legs. Professor Vulpian scatters to the winds the fanciful hypothesis so popular in some quarters, and which attributes so much and varied influence to disturbed circulation in the cord. Experimentally and theoretically, he demolishes that "deus ex machinâ" of the pathologist in trouble—"vasomotor changes"—and proves his assumptions to be mere "post hocs." Congestion of the cord may, however, produce symptoms in certain morbid conditions; and he describes a case of suspected varices of the organ where ergotine was of great service. With reference to anæmia, it is impossible to conceive the occurrence in the human subject (except perhaps through a sudden diminution of atmospheric pressure) of a paralysis similar to that produced in animals by artificial embolisms of the spinal vessels; and unless with Hammond we believe in the possibility of anæmia of individual bundles of fibres, how are we to explain the

persistence of sensation in those cases where paraplegia has been attributed to vasomotor spasm?

We rapidly pass over several topics which the author discusses ably, but without adding much to our previous information, noticing only on the way the importance he attributes to the rich nervous supply of the pia mater and arachnoid, especially on the posterior aspect of these membranes. These facts may explain not only the pains of arachno-lepto-meningitis, but also perhaps the greater intensity of the inflammatory process generally prevalent in the posterior surface of the cord. Patches of chronic arachnitis often found post mortem may, he thinks, explain certain obscure spinal symptoms; but, notwithstanding his efforts, he has not been able to trace very clear conclusions as to their clinical import.

Professor Vulpian has been the first to establish a distinction between the diffuse and the systematic forms of myelitis, and to explain their pathogeny by assuming (a view now very generally adopted by French writers) the former to consist in a primarily interstitial, the latter in a primarily parenchymatous inflammation. Among the usual causes of acute diffuse myelitis are propagation (from meningitis), traumatism, tumours, fatigue. He fully discusses the ætiological bearings of cold. It is clear that since meningitis is as frequently produced by exposure to cold as myelitis, we cannot assume in the latter a direct influence from the periphery through the afferent nerves. There must be a reflex action transmitted through the sensory (trophic) nerves supplying the meninges and the connective tissue of the cord itself. The impression made on the surface disturbs the nutritive functions of the centres with which the part is in relation, and this is manifested by an inflammatory process in the tissues supplied by the same centres.

What individual tissue or organ will become the seat of the morbid change depends, of course, upon some idiosyncrasy or special predisposition. The theory is applicable not only to myelitis, but to pleurisy, peritonitis, nephritis, &c.—in a word, to all inflammations due to the effects of a chill to the surface. Experiments show how readily myelitis can be induced by irritation of peripheral nerves; and here we have a satisfactory explanation of the so-called reflex paralysis and

atrophies, according to the author. The administration of lead and bromide of potassium to dogs has also been followed with marked meningitis. We pass over the discussion of acute diseases in their relation to myelitis, as we have had lately the opportunity of reviewing Dr. Laudouzy's exhaustive treatise ('BRAIN,' X.). We need not say anything of the pathological anatomy and of the sensory and motor symptoms of myelitis, as the author does not contribute any new facts with reference to them. The vasomotor centres may be excited or paralysed. In the first case, should the constriction of the vessels extend to the venules, the limbs become cold and pallid; if confined to the arterioles they are cold and livid—precisely what we find obtaining in Raynaud's asphyxia of the extremities. Paralysis of the vasomotors is characterised by redness and heat at first, followed by blueness and coldness due to the increased cutaneous radiation and the atrophy of the surrounding tissues. Among the trophic disturbances the author discusses the "acute decubitus" (a misnomer he thinks), which he attributes to the united effects of the several causes at work, and not to a specific action of the disturbed innervation. The cystitis, which is often present, is explainable on similar principles. The innervation of the lungs, stomach, intestines, &c., if disturbed, may set up various morbid processes in these organs.

The intensity, extent, and localisation of the myelitic process vary greatly; and to these variations correspond all the grades of the disease, from the apoplectiform type, which kills outright, to the lightest type, which betrays itself best by the slight paresis of a limb. The process, again, may attack the whole cord "d'emblée," or come under the designation of ascending or descending. Professor Vulpian incidentally draws a parallel between Landry's disease and acute ascending myelitis, and thinks that whilst there is as yet no pathological reason for identifying them, neither is there any ground for explaining the former by a toxæmic process: the question is still open. It must not be forgotten that several cases, recorded under the name of Landry's disease, are really nothing but more or less aberrant forms of myelitis, as the description of the symptoms clearly shows.

Among the various forms of chronic diffuse myelitis, we must first distinguish those in which the white matter is affected from those in which the grey matter is the seat of the morbid process; in other words, leuco-myelitis from polio-myelitis. Leuco-myelitis, again, may be cortical or annular, as in many cases secondary to meningitis, where a ring of white matter only is affected (mainly on the posterior aspect, where we have seen the meningitic process to be most intense), or spreads through the whole depth of the white columns, as is typically the case in general progressive paralysis. The pathological change in the connective tissue is the same in diffuse and in systematic myelites; but in the former the destruction of nerve fibres is very rarely as complete as in the latter, where, as above mentioned, a parenchymatous inflammation is assumed to be the primitive lesion by the French school of neurology.

In chronic diffuse polio-myelitis the alterations may be confined to the peri-ependymary region, a form which has been carefully described by Hallopeau as "peri-ependymary myelitis," and is characterised by the development of vacuoles, often filled with liquid. Michaud has stated that the lesion of tetanus is allied to that obtaining in this form of myelitis; and Professor Vulpian, in his experiments on the toxic effects of large doses of bromide of potassium, has found the paralysis of his dogs apparently arising from a similar peri-ependymitis.

True diffuse myelitis affects both the grey and the white matter, though not evenly, perhaps, nor universally. The symptoms of it will, of course, vary with the mode and intensity of its progression; and its diagnosis will be based upon the combination of the symptoms present, motor, sensory, trophic, reflex, &c., which will also frequently reveal the localisation of the pathological alteration in the cord. Duchenne has committed a serious error in separating under the name of "general subacute spinal paralysis" certain cases of diffuse myelitis, and giving them a rank co-equal to the anterior poliomyelitis he has defined with such masterly precision.

The second half of Professor Vulpian's volume deals with progressive motor ataxy, as the chief variety of systematic myelitis has unfortunately been named. Since 1862 our author has pleaded for the syphilitic etiology of tabes;

out of 20 patients 15 at least have had syphilis, according to him. Fournier (1876) gives a still higher percentage, 80 per cent. Gowers ('Diseases of the Spinal Cord,' p. 62), in a curiously-expressed passage, in which he does not mention these and other earlier observations, has espoused the same view. In a paper published in 1879 he "expressed the opinion that one-half of the cases there is a history of antecedent syphilis. The same opinion has since been expressed by Erb," &c., and he states that out of 16 patients lately examined by him 11 had had chancres, and many symptoms of constitutional syphilis. Against the actual causal nexus between syphilis and tabes many arguments can be urged. First, the discrepancy in the statistics of a large number of competent pathologists, who have not found a high proportion of specific antecedents to tabes (Westphal, for instance, who gives a much lower figure; and, besides, on the ground of numerous post-mortem examinations, doubts the syphilitic nature of the disease). Leyden rejects the theory altogether. Chauvet plainly says (1880), "Syphilis never will cause a primitive sclerosis of the posterior radicular zones." Again, it has been stated by Westphal, Bernhardt, and others, that syphilitic antecedents have never been traced by them in any female case of tabes. And yet would not the prevalence of the infection among prostitutes make them peculiarly liable to that manifestation of the disease? Finally, whatever may be said to the contrary, the utter failure of mercury and iodide in the treatment of tabes[1] is a presumption against the syphilitic nature of it. Do we not, in fact, look upon the successful administration of these drugs to patients suffering from symptoms of doubtful origin as an almost diagnostic sign? It would be strange indeed that tabes should make such a startling exception to the rule. All we are disposed to concede is that the syphilitic cachexia may increase the natural predisposition of certain individuals

[1] In a report of a paper read by Dr. Dowse ('Brit. Med. Journ.' Dec. 11) he is made to say that "every case of locomotor ataxis, with very few exceptions, may be traced to syphilis as a cause; and that every case of the disease was curable" if treated sufficiently early and vigorously. Happy doctor, happy patients! But we are at a loss to understand the rationale of this anti-syphilitic treatment: Bromide, ergot, counter-irritation, besides merc. bichlor. gr. ¼ per diem. Nerve-stretching appears to be the most promising form of treatment. Its success would be a fatal argument against the specific nature of the disease.

to tabes as it does to other intercurrent affections. We should remember, also, how the once universal idea of the relationship between tabes and sexual excesses has been shown to be fallacious. Another point to be noted is the author's opinion that there is a relation of cause and effect between hysteria and tabes in women. Would not rather both be best considered as manifestations of a neurotic tendency? A more important connexion is that of tabes with progressive general paralysis. Professor Vulpian gives a very full symptomatology, with copious illustrations, by means of well-selected and described cases, of the three periods of tabes. He lays special stress upon the invariable presence of sensory troubles; some anæsthesia is the rule; but the perception of cold is often singularly vivid and calls forth reflex movements. A full discussion of this part of the work, however, as well as that which treats of the pathological alterations, would lead us beyond our limits. Moreover, the author does little more than give an exposition of the already known facts. The last two lectures, however, deal with the pathogeny of the morbid phenomena, and deserve to arrest our attention for a while. The author is strongly of opinion that the nature of the primitive lesions is not a pure atrophy, but depends upon a morbid irritation or an inflammation of the axes-cylinders of the posterior fibres. The atrophy so widely prevalent among the affected parts is secondary. Some authors have propounded a doctrine opposed to this view. According to them the sympathetic was primarily affected; and upon this lesion followed some disturbances of the nutritive vessels of the cord, to which again the atrophy of the nervous elements was secondary. Unfortunately for this theory its first link is weak: the sympathetic has often been found absolutely healthy in advanced cases of posterior leucomyelitis. But could not the disease be secondary to vascular changes setting up an intestinal myelitis? This is a more insidious argument; but it must be remembered that the posterior roots are atrophic also. How could this be accounted for upon this hypothesis?

The author develops his views with reference to the primitive morbid irritation, probably of inflammatory nature, of the axes-cylinders, with great skill and force. The secondary

lesions of the connective tissue are accounted for in a most ingenious manner. The axes-cylinders lose their vitality, and act to all intents and purposes as foreign bodies, or sequestra, and stimulate the surrounding tissues to an inflammatory reaction. Passing to the order of succession in which the lesions spread, and having explained how the inflammatory process set up in the adneural structures may be propagated to the meninges, Prof. Vulpian shows that the accepted theory of Charcot and Pierret does not remove all the difficulties. For how does the morbid process pass from the fibres of the external radicular zones, first to the rest of the posterior fasciculi, and second, to the posterior roots? If there be any fact well established in physiology, it is Waller's discovery of the trophic dependence of the posterior spinal fibres upon the posterior root-ganglia. Though unable to reconcile the conflicting data, the author visibly inclines to place in the posterior roots the starting-point of the morbid process. In an ascending direction sclerosed tracts may be followed throughout Goll's columns, which have undergone secondary change.

Whatever difficulties we meet in our attempts at an explanation of the various symptoms and their anatomical substratum, they vanish in presence of the impossibility of accounting for the pathogeny of that substratum itself. Clinical and experimental investigation here completely fail us.

It is commonly thought that the anæsthesia usually prevalent in tabes depends upon the sclerosis of the posterior columns. Schiff's experiments are relied upon as a physiological basis. But these are far from being above criticism, and Prof. Vulpian has failed to obtain the same results as the Genevese observer. Not only was no anæsthesia noted after section of these columns, but there supervened hyperæsthesia (as well as hyperalgesia). Hence it is only when the posterior roots are involved, that sensation can be diminished in posterior leuco-myelitis.

How is it that the tactile sensibility of a whole limb may be lost, whilst a number of sound fibres exist in the posterior roots? Are we then to conclude, to a special set of fibres for each quality of sensation—of touch, of temperature, of pain, &c.?

No, but simply accept the hypothesis (borne out by the fact, that more fibres enter the posterior ganglia from the periphery than leave it to enter the cord), that a sensation carried to the ganglion by one fibre is there spread over (so to speak) the whole root. Any deficiency in the number of fibres here will, therefore, diminish the intensity of the sensation. With reference to analgesia, it is only anæsthesia carried one degree further; pain, too, is but sensation carried one degree further. Painful anæsthesia is simply due to the psychical localisation in an anæsthetic limb of pains due to an irritative process of its central sensory tract.

The author incidentally discusses the anodyne effect of morphia injections along the course of the nerve to which the painful sensations are referred, such as the characteristic lightning pains. He attributes it less to the general absorption of the drug than to the centripetal effects of the local anæsthesia induced by the injection. In the same way local applications of mustard, chloroform, &c., allay the shooting-pains, not by reflex central vasomotor changes, as is so often presumed, but by some direct excitation of the ganglia.

In trying to explain these facts, as well as the various sensory disturbances common in tabes, we must remember two facts, that the fibres from the periphery to the ganglia are usually healthy, and that they have probably no direct continuation through the ganglia to the cord, but are interrupted there, the further conduction of impressions requiring possibly the integrity of the whole root. When, therefore, the number of fibres constituting that root is diminished, powerful and protracted stimulations may yet be felt when all weaker and shorter ones fail to be perceived. Central and peripheral anastomoses may also be brought in to explain the reference of sensations to wrong spots on the periphery. The author tries to explain the persistence, or even increase, of thermæsthesia by the special mode of central stimulation, which we must assume to be the condition of the perception of heat and cold when the existence of special fibres is denied. We know how the sensory tracts of the cord are sometimes stimulated by the passage of a hot or cold sponge along the vertebræ: hot and cold impressions, therefore, seem to have a peculiar

power of stimulating the spinal sensory elements under certain morbid conditions. With reference to the retardation of transmission of sensations (a phenomenon, by the way, the frequency of which we believe to be greatly exaggerated by some writers), it is not due to the diseased condition of the posterior cords, for it is not observed in animals on which these tracts have been destroyed, but rather to the diseased condition of the posterior ganglia, where, as above mentioned, the different fibres are probably interrupted. The posterior grey spinal matter, which is assumed to be æsthesodic, does not generally present sufficient signs of alteration to account for the delay.

It is rare that absolute anæsthesia prevails, even in advanced cases. Protracted and energetic faradisation often succeeds in restoring a limited and temporary sensibility at the spot where it is practised.

The last few pages of the book are devoted to the subject of the motor ataxia. It is unfortunate that more space was not devoted to this interesting and important subject. As everybody knows, two leading hypotheses occupy the field. The first assumes the disordered balance of muscular action to depend upon the want of co-ordinated spinal stimulus, due to the lesion of the intercellular fibres assumed to exist in the posterior columns; the second explains it by the impaired impressions from the periphery, and the deficient knowledge of the whereabouts of the limb, so to speak. In the first instance, the impulses are correctly given, but incorrectly obeyed; in the second they are incorrectly given, owing to mistaken information. To discuss these points would carry us far beyond our present limits. Prof. Vulpian accepts the second theory (known in Germany as that of Leyden), though open to very grave objections. His reason for rejecting the first is simply that the existence of the intercellular fibres has never been demonstrated anatomically. Here, as all through the book, we are made to feel the utter neglect with which too many French pathologists and physiologists treat foreign, and more especially German, science. No doubt the modern school of French neurology is shedding splendid rays of light in every direction. But there are luminaries of the first

order elsewhere, and completeness is a quality as well as originality. Could not M. Vulpian combine the former with the latter to a greater degree? He often quotes Duchenne and Pierret; why not add the names of Erb and Flechsig? Has not electro-diagnosis been raised by the former to a far more dignified position than that where Duchenne had placed it? and have not Flechsig's researches on the development of the cord opened a new era in its physiology and pathology? Yet in spite of these deficiencies Prof. Vulpian's last work remains fully worthy of its author's great reputation.

A. DE WATTEVILLE.

The Science and Practice of Medicine. By WILLIAM AITKEN, M.D. Edin., F.R.S. Seventh Edition. London: Charles Griffin & Co., 1880.

A BOOK on the Science and Practice of Medicine which extends to 2381 pages of unusual amplitude, and has gone through seven editions in twenty-three years, is beyond the reach of criticism in a double sense. It baffles it, by its comprehensive immensity, and it is independent of it by its assured reputation. We feel that it would be impertinent to do more than welcome Dr. Aitken's great and standard work in its new, enlarged, and remodelled form. Excellent from the beginning and improved in each successive issue, this work has now, with vast and judicious labour, been brought abreast of every recent advance in scientific medicine and the healing art, and affords to the student and practitioner a store of knowledge and guidance of altogether inestimable value.

It is in those sections of Dr. Aitken's book that are cognate to the scheme of this journal, that the most important alterations and extensive additions have been made in the new edition. Undismayed by the magnitude of the task, the author has vigorously grappled with the immense and confused crowd of facts bearing on diseases of the brain and nervous system, that have been brought to light of late years, and has succeeded marvellously in reducing them to order, and in assigning to them their just rank and station in the

body of previously ascertained knowledge. So comprehensive and accurate is the survey taken of this department of medicine, that the first 530 pages of the second volume in which it is comprised would, if printed separately, form perhaps the best text-book in our language for the student of neurology and insanity. A masterly and philosophical review, characterised by the precision of the specialist and the breadth of the catholic physician, is presented in those pages of the varied phenomena connected with morbid conditions of the nervous system in their relations with anatomical structure, chemical composition, physiological uses and pathological changes. An introductory sketch of the direction taken by modern inquiry into the functions and diseases of the nervous system, is followed by a general outline of the crude anatomical constituents of the nervous system, and by a detailed account of the histology and arrangement of the membranes of the brain and cord, and of the specific gravity of the brain, and its weight at different ages and in different races and conditions. Next comes a description of the external configuration of the brain, of its several convolutions and minute structure, and of the composition and structure of the other encephalic centres, as complete and minute as is to be found in any handbook of anatomy. The succeeding sections deal with the vascular supply and chemical properties of the brain and nervous system, and then follows a truly admirable summary of the physiological relations and functions of the several parts of the brain and nervous system. Here the labours of Flourens, Laycock, Luys, Van der Kolk, Goltz, Hughlings-Jackson, Hitzig, Ferrier, Purkinje, Longet, McKendrick, Robertson, and many other observers and investigators are epitomised and digested with great ability and discrimination, and all that has been established with regard to localisation is clearly brought out. From physiology Dr. Aitken passes to morbid anatomy, and discusses the pathological relations of the several parts of the brain and nervous system, furnishing guides to the clinical investigation and diagnosis of disease. The laws of isolated conduction of sympathy and irradiation and of mental manifestations are explained, and the localisation of disease in the brain, spinal cord

and nerve trunks, is set forth as explicitly as our present state of knowledge warrants. Passing from general and preliminary considerations to the concrete and actual examination of disease, Dr. Aitken introduces first functional disorders, and secondly substantive diseases of the brain and nervous system. Neuralgia, cephalalgia, anæsthesia, labyrinthine vertigo, infantile and puerperal convulsions, scrivener's palsy, largysmus stridulus, paralysis agitans, chorea, hysteria, rhythmical chorea, epilepsy, catalepsy, tetanus, hydrophobia, sunstroke, general paralysis, hemiplegia, bulbar paralysis, paraplegia, spinal hemiplegia, polio-myelitis, progressive muscular atrophy, pseudo-hypertrophic paralysis, locomotor ataxy, local palsies, delirium, pachymeningitis, cerebral and spinal meningitis, tubercular meningitis, chronic hydrocephalus, encephalic apoplexy, softening of the brain, encephalic tumours, disseminated sclerosis, aphasia and insanity are in turn and exhaustively considered, besides many subsidiary topics which we have not enumerated. Each disease is dealt with in all its bearings, in a scholarly, systematic, and practical way, a definition being attempted in each, and all that is known of symptomatology and pathology being ably expounded. The sections on epilepsy and insanity are the most elaborate, and mount indeed to the merit of tolerably complete treatises on those subjects. The medico-legal relations of insanity are not overlooked, nor are the relations of states of the eye to diseases of the nervous system, a chapter being added on that topic.

If we were to find any fault with Dr. Aitken's 'Science and Practice of Medicine' it would be to the effect that the directions for treatment which it supplies are not always as full and minute as could be desired. As medicine advances and becomes more scientific—and that it is doing so Dr. Aitken's book is a most signal proof—treatment emerges from empirical simplicity into rational complexity. The means used become more simple—prodigious concoctions giving place to single remedies—but the considerations affecting the use of these means become infinitely more intricate and numerous. Determined and regulated no longer by an all-embracing theory, a name or a rule of thumb, but by a better appreciation of the causes of disease and a juster

recognition of the disordered processes and material changes which form its constituents, treatment has branched out into a manifold heterogeneity, requiring a minuteness of description that was formerly unnecessary and unknown. Enlarged by the discovery of new remedies and by the new applications of old ones, and modified from time to time in each case in accordance with the variations of symptoms which modern observation keeps under close and incessant supervision, the treatment of disease has become laborious, and demands in many instances a minute observance and resource, scarcely inferior to what is required in Listerism as applied to surgical wounds. Of course Dr. Aitken is alive to all this, and has under many diseases entered fully and explicitly into the whole duty of the medical attendant, from first to last, tracing out the parallelism which should be observed between treatment and the progress of morbid processes, and supplying prescriptions which have been well selected, and must prove eminently useful to the busy practitioner. Nothing could be better than the directions he gives for the treatment of megrim, of epilepsy, of tetanus, and of neuralgia. But under other headings his account of treatment and recommendations regarding it are occasionally somewhat meagre. Thus the treatment of catalepsy is dismissed in half-a-dozen lines; that of locomotor ataxia in half a page, and that of insanity, which is considered as a whole and not in relation to the many distinct varieties of mental aberration, which require distinct courses of medical and moral management, in five pages. In a few cases, too, we find no mention made of remedies which are believed to be of real value. Thus nitrite of amyl is not alluded to under the treatment of angina pectoris, nor Calabar bean under that of general paralysis, nor hyoscyamine, conium, chloral, nor arsenic, under that of insanity. Such omissions, however, are few and far between, and can only be discovered by careful search, and it would be ungracious to refer to them save with the desire that every flaw, however insignificant, should be removed from a classical work, which does honour to British Medicine, and is a compendium of sound knowledge.

J. Crichton-Browne, M.D.

Clinical Cases.

A CASE OF EPILEPSY:—AURA OF TRUE VERTIGO — EMOTIONS OF ANGER AND REMORSE — VAGUE COMPLEX REMINISCENCES — RIGHT-SIDED SPASM—FAMILY HISTORY.

BY CHARLES MERCIER, M.B. (LOND.), F.R.C.S.

Senior Assistant Medical Officer at Leavesden Asylum.

Mary H., æt. 36, unmarried. Family history from patient, who is fairly intelligent. Her mother used to have fits, in which she screamed, fell, and used to lay hold of things. On one occasion she fell into the fire and took hold of the bars, burning her hands severely. Mary H. had two sisters, of whom the elder had severe fits, in one of which she died. The other had fits until puberty, at which time they ceased. This second sister was badly frightened one day, was brought home, and died the same evening, but whether in a fit or not the patient cannot say. The mother had no brothers or sisters. The father died "asthmatical."

In obtaining the following account, the utmost caution was exercised to put no questions that should in the least degree suggest the answer, and the description of her sensations is taken down as nearly as possible in her own words.

She has both "fits" and "sensations," which both begin in the same way. They come on with a giddiness. She feels very trembling, and the place and everything seems as if it were really going round before her. Subsequently she says that everything seems to be dancing and going all manner of ways; but on being told to move her hand in the direction in which things appear to go, she waves her right hand with a circular motion from left to right, and adheres to this description when severely cross-examined. This continues for a "good while," and then is followed by a feeling "as if she must cry out," "as if she must give a good cry and hold on to some one." This is because she feels the fit coming on, and wants to hold on to people, so that they can take care of her

and see that she doesn't fall, and doesn't do anything wrong. On further questioning she says that she catches hold of people because her head feels queer, and because she is afraid of doing anything wrong. She repeats many times in the course of her examination that she hopes she does nothing wrong in the fits, that she doesn't mean to do anything wrong, and that as soon as the fit is over she always asks what she has done, and feels very sorry for having done wrong and for having said anything that she ought not to say. As a fact, however, she neither uses bad language nor is she violent during the attack, nor does she ever express regret after it, as she imagines that she does. Besides this she says, "Before the fits I think very much of where I have been (long ago) and how they might be, and I should like to be with them again, all manner of things." Again, "I feel as if I should like to go to them, and say to them, 'I should like to do my duty to you and work with you, and be happy with you.'" The "they" refers to her sister and her sister's family, with whom she once lived. These feelings occur after the giddiness, before the "fit," but at the end of the "sensations." To this statement she adheres without wavering when questioned on different days. Before the "sensations" she feels "workified." "As if I wanted to be working around and around and doing things." *After* the fits she has the same feeling. Again, she says, after the fits she feels as if she were going to hit people. She feels towards them as if they had done something at her. She feels that she would like to hit them in order to vent her spite on them—to be revenged on them. These feelings towards other people she does not experience at all in the sensations.

While examining this patient, I was called away to observe another who was in a fit at about six yards' distance. While observing this second patient my attention was attracted by a harsh cry as of some one in terror, and turning round I saw Mary H. sitting in the chair which I had just quitted and still emitting the cry. I immediately went to her, and on my approach she rose from the chair and stood beside it, with a grieved and anxious expression of face. The whole affair passed so quickly that it is not possible to particularise further than to say that this is the impression that her face gave me. It is certain that her "grief muscles" were strongly in action. On immediately asking the nurse who was standing beside her during the attack to which side her head was turned at the outset of the fit, she answered, "To the right"; and it may therefore be taken for certain that this was correct. On then at once asking the patient how she felt, she answered, "I feel better now. I feel as if I want to go to work—

to be doing something." She also said in answer to questions that she knew she had just had a fit. On the following day, however, she was ignorant of the whole affair, and of my asking her about it. The duration was between five and ten seconds.

Nurse's Report.—Mary H. has both "sensations" and "fits." The "sensations" are very frequent—one or two every day. The first thing noticed is that she gives a slight scream and makes for a chair. There is generally time to get her into one, and if she did not reach one she would fall. She "works about," and has to be held (to prevent her from falling) for a few minutes (probably moments). Then she becomes very restless and begins to undress herself. The nurse who has witnessed these "sensations" daily for four years, has never known her catch hold of people either in or after the "sensations," neither has the patient ever attacked people or been known to look angry. As soon as the restless movements cease she gets up and goes to work.

The "fits" come on sufficiently deliberately for the fall to be prevented, or she would fall. Judged by the standard of the other ninety-nine epileptics under the charge of this nurse, the fits are rather severe; they affect both sides, and are followed by "deep sleep," and as soon as that is over she gets up and goes to work. These severer attacks occur at intervals of three or four weeks. At longer intervals—of three or four months—she has batches of fits, after which she is alienated for a short time, strips herself naked, and runs up and down the ward screaming.

Remarks.—The importance of this case in regard to the speculations of Dr. Hughlings-Jackson in the last number of 'Brain' is manifest. The under-mentioned are the points on which it bears directly. The epileptic attack, beginning with true vertigo, is associated with right-sided spasm, and objects appear to be displaced to the right. This is in harmony with Dr. Jackson's supposition. 2nd. There was after the fit no sign of aphasia of any degree, i. e. no using of wrong words or approximate expressions even. 3rd. The vague complex reminiscence occurred in association with right-sided spasm at the outset. 4th. This intellectual state occurred in association with "the objective warning of vertigo." In the last three particulars the occurrences in this case are not in harmony with Dr. Jackson's speculations. It should be added that the patient is right-handed.

The case is important also as illustrating other points in the natural history of Epilepsy.

The identity of the feelings experienced during the "sensation" with those experienced at the outset of the "fit" is

proof, if proof were needed, of the identity of nature between *petit mal* and *grand mal* with universal convulsion. It shows that the discharge in the latter begins in the same region and spreads in the same manner as in the former, and that the difference between the two forms of seizure depends mainly upon the greater or less extension of the discharge. Whether the rather elaborate mental states occur during and in consequence of the discharge itself, or whether they occur from over-activity of regions from which control is removed in consequence of the discharge in regions above them, is immaterial to the present issue. Whatever the *modus*, the same physical state must accompany the same mental state.

Another important fact is that what she calls the "workified feeling," "as if I wanted to be working around and around, and doing something," occurs before the sensation and after both the sensation and the fit. In other words, the same mental state, and therefore the same physical process in the brain, occurs early in the onset and late in the recovery, corroborating and illustrating the doctrine that the regions of the brain resume their functions in the inverse order of that in which they were discharged; and that this law obtains as truly in those higher and less deeply organised regions of the brain whose disablement corresponds with loss of consciousness, as in those lower and more deeply organised regions whose disablement corresponds with hemiplegia.

ON HEMIPLEGIA AND HEMIANÆSTHESIA IN AN IDIOT BOY, AS THE RESULT OF PARALYSIS OF THE LEFT CEREBRAL HEMISPHERE, FOLLOWING A BLOW ON THE HEAD.

BY DONALD FRASER, M.D., PAISLEY,

Visiting Physician, Riccartsbar Asylum.

So far as my experience and reading are concerned, the case which I am about to relate is, in some respects, unique. But for the opportunity of making a post-mortem examination, the symptoms would have been considered as indicating plainly some such organic lesion as a considerable effusion of blood affecting the left hemisphere; while as it was, the most careful examination failed to show any gross lesion. The case has further some special interest at present, when so much attention is being paid to the subject of hemianæsthesia.

W. C., aged 11½ years, of illegitimate birth and the subject of congenital idiocy, was admitted into Riccartsbar Asylum, on Jan. 20th, 1878, in good bodily health. He was a chubby-faced active little fellow, about 4 ft. in height. He walked with a very slight inclination forwards, and carried his right arm awkwardly, or as if he had not quite the same control over it as he had over his left; and when he was allowed to feed himself, which was not often, he used his left hand. He was readily attracted by a bright object, and was often troublesome in his attempts to examine such objects as one's watch guard. His intelligence was very low. His speech was confined to a few words, used apparently without reference to their meaning. These words consisted of names which he must have been in the habit of constantly hearing before his admission to the asylum. "Granny," for instance, was often uttered by him. Sight and hearing were apparently normal. He was wet and dirty in his habits. His right testicle was absent.

His head measured in circumference	$19\frac{5}{8}$	inches
Antero-posterior diameter taken from root of nose to occipital prominence	$12\frac{1}{2}$	„
Distance from the one meatus to the other over vertex . . .	$5\frac{5}{8}$	„
Distance from the centre of forehead to meatus	$5\frac{1}{4}$	„

He was reported to have had a severe illness about a year ago before his admission, which was marked by head symptoms, and preceded by a fit. It was also reported by his friends that he was in the habit of taking a "fit" about once a year or so, though during his stay of ten months in the asylum he was free from epilepsy.

On the 30th Nov. 1878, he was thrown down by one of his fellow patients, so that his head struck the floor of the day room with some force, though it was not sufficient to bruise the scalp, or produce any other external mark of injury. He appeared to be none the worse immediately after the accident, but in about 15 or 20 minutes was found by the head male attendant lying on the floor, in what he, the attendant, described as a fit, which was attended by pallor of the face and vomiting. A few hours later he was unconscious. His condition next morning, twenty-four hours after the accident, was as follows: His head and eyes were turned to the left side, his right eye most so. The pupils were dilated, but reacted to light. The whole of the right side of the body, including the right side of the face, was anæsthetic, while the right arm and leg were paralysed. Tickling the sole of the right foot caused a scarcely perceptible reflex movement. A pin thrust into various parts of the right arm and leg was followed by no action whatever, but when applied to the left caused shrinking and withdrawal of the limb. The temperature in the axilla was 102°. Respirations were 18, and pulse was 84. His bowels were opened freely during the day by means of an enema. Beef tea and milk, when given to him, were swallowed easily. Next day, the 2nd Dec., his pulse rose to 90, but the breathing continued quiet and regular; egg and milk mixture was given, and swallowed without difficulty. The paralysis of sensation and motion on the right side was still complete, otherwise he lay like one in a profound sleep. On the third a blister was applied to the vertex. 5th Dec., pulse 90, respiration 16. The conjunctivæ of both eyes were injected, the right particularly so, while the lower third or more of its cornea was muddy and soft-looking. 6th Dec., he was so far conscious that he picked up with his left hand the tobacco-box of a fellow patient when it was put within his reach. He now presented a very striking appearance. His right limbs were limp and motionless, the paralysis of motion

being still profound; the cornea of the right eye, so far as it was uncovered with the lid, was white and ulcerated. The breathing was quiet and regular. His consciousness was so far restored that on his attention being called to the tobacco-box which was held near his left hand, he slowly and in a dreamy way grasped it, and kept hold of it. Pricking the left arm and leg was followed by expressions of pain and movements of the limb. This improvement did not continue, for on the next day his pulse rose to 120°; his temperature being $98\frac{1}{5}$°. The tobacco-box now required to be put into his hand, which closed over it slowly as that of a sleeping child might do. He still breathed quietly and regularly as if in a deep sleep.

On the 9th Dec. pulse 140, respirations 20. The left cornea now appeared softened in its lower third.

10th Dec. The left side was now found to be hemiplegic, and he was evidently sinking. He died next day at 11 A.M.

A post-mortem examination of the body was made about 30 hours after death by Dr. Richmond Paisley and myself. We were assisted by Dr. Cunningham, now of Alva, who had seen the patient with me during life. There were no external marks of injury on the body. The scalp and skull were carefully examined, but shewed no evidence of injury. The thoracic and abdominal organs presented no appearance calling for remark. The calvarium was of medium thickness, and the dura mater was slightly adherent to it in several places. While the brain was still in situ the left hemisphere was seen to be fuller or larger than the right. The superficial vessels of the dura-mater were full and somewhat hyperæmic. The subarachnoid fluid was rather more abundant than usual. On removal of the brain the left parietal lobe was seen to be distinctly larger than the right. There was at the same time a marked depression of the orbital surface of the left frontal lobe. The optic thalamus and corpus striatum on the left side were also unmistakeably larger than those on the right. A desire to preserve the relation of the different parts prevented me from weighing them. Very careful incisions were made, particularly in the region of the left optic thalamus and internal capsule, but no lesion of any kind could be discovered. The convolutions appeared normal in arrangement and number and were deep, while the grey matter seemed deeper than usual. The brain weighed as a whole $32\frac{3}{4}$ oz. It was set aside, the relations of the various parts being carefully maintained, and submitted to a second examination by Dr. Joseph Coats, Pathologist to the Western Infirmary, Glasgow, who was also unable to detect any lesion of it or its membranes.

The spinal cord was examined and found normal, and por-

tions of it were also hardened in chromic acid. Sections presented nothing unusual when examined microscopically.

So far as the weight of this brain was concerned, it was only a few ounces below the average weight of the brain at his age. The obvious departure from the normal condition was the unequal size of the hemispheres and central ganglia. The greater size of the left hemisphere, or more strictly the left parietal lobe, was not here, as is often or generally the case where such a symmetry is found, due to atrophy of the right, for the latter was functionally the more perfect of the two. Dr. Crichton-Browne[1] refers to a pronounced excess in weight of the left over the right parietal lobe in both sexes, amounting in men to 8·1 grammes, and in women to 4·8 grammes, and considers this difference to be due to the higher development and education of the left hemisphere, which presides over the more specialised and voluntary motor performances of the right side of the body. In the brain under consideration, the excess in size and presumably in weight of the left parietal lobe over the right was so considerable that it at once caught the eye; yet the right side of the body was here the least capable of voluntary motor performances.

Mere inequality in size of the two hemispheres is of itself, then, compatible with the vigorous and normal performance of the cerebral functions. Yet, according to Griesinger, inequality of the hemispheres is frequently observed in the brains of idiots, where, however, it is obviously associated with pathological conditions. The apparently greater depth of the cortical layer in this brain was in keeping with another peculiarity referred to by Griesinger,[2] that of an unusual abundance of grey substance in the brains of some idiots. He even refers to the grey matter in the brains of certain idiots as being "greater than the white." The most likely explanation of the nature of this condition, particularly where the excess is so great as that referred to by Griesinger, is that the brains in question were in an undeveloped condition; for, according to Meynert,[3] in the cerebral medulla of "the fully developed brain no finely granular substance appears to be present, but during the period of development it exists, and probably occasions the *more grey than medullary aspect of the immature organ.*"

Only a complete microscopical examination of the component parts of such a brain as we are discussing would be of any great value. Apart from this, I examined numerous

[1] 'On the Weight of the Brain and its component parts in the insane.' Brain, Vol. II. p. 65.

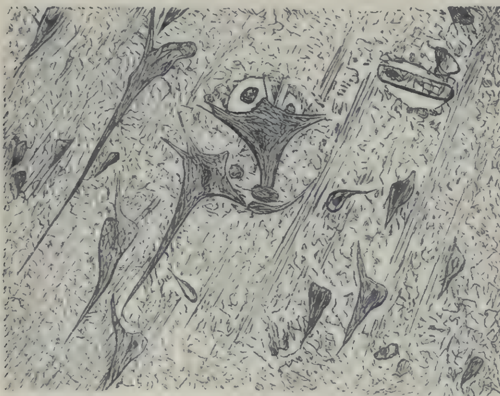
[2] Griesinger 'On Mental Disease,' p. 360.

[3] The 'Brain of Mammals,' by Theo. Meynert, p. 390. Human and Comp. Anat., New Syd. Soc. Trans.

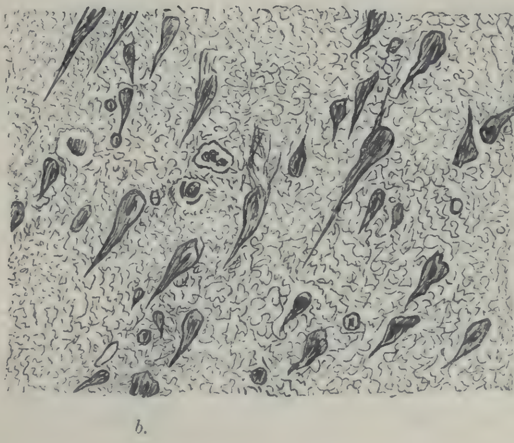
sections, made chiefly from pieces of the left cerebral hemisphere. I have satisfied myself, as the result of repeated examination, that the pyramidal cells, particularly of the second, and to some extent of the third, layer of the cortex indicate a condition of defective development. The processes are fewer than normal; indeed, the apical process is, in most instances, the only one present. The cells are, as a whole, more ovoid in shape than is usual, and many have what might be described as a somewhat withered appearance. It is necessary to speak with some degree of caution on this point when dealing with hardened specimens. To some extent they approached in character to the description which Dr. Bevan Lewis, in 'BRAIN,' Vol. II. p. 364, gives of the appearance of these structures in such epileptic idiots' brains as he had examined in the fresh state, though much of what he describes could only be observed in that state. The sketches *a* and *b*, magnified about 300 diameters, made with the aid of the camera lucida, give a very accurate representation of a specimen where, if anything, the appearances were more normal than in many others.

a represents a group of well-marked large pyramidal cells of the inner portion of the third layer, specially selected because of their large size and normal appearance. They also serve to show, in contrast to *b*, that the angular or pyramidal shape is not necessarily affected by the hardening agent. In *b* the general appearance of the larger cells of the second layer is well represented.

There can be little doubt that in this case the hemiplegia and hemianæsthesia were due to changes affecting the cortex of a large portion, if not the whole, of the left hemisphere, and influencing more particularly its cells. This boy having an unstable and unsymmetrical brain, with at least its cells imperfectly developed, this condition being most marked in the left hemisphere, suffers as above described from the effects of a blow on the head: a blow which in a healthy child might have given rise to the symptoms of slight shock; or at most to what I have occasionally seen from such a fall, a slight epileptiform seizure, succeeded immediately by involuntary emission of urine and a sleep from which the child awakes well. Here indeed the paralysism was indeed preceded by a fit, but instead of passing off it continued, and was also more profound than is usually seen in post-epileptic paralyses. So much was this the case that, as we have seen, ulceration of the lower half of the right cornea took place, the portion, by the way, which was most exposed to external causes of irritation. It was only during the last two days of his life that the right hemisphere began to indicate an equally serious failure



a.



b.

To face p. 540.

in its functions. During the first few days it showed signs of slight irritation. At least to this I partly attribute the forcible turning away of the face and eyes from the paralysed side, although no doubt the usual explanation of this symptom—that it is due to the muscles of the sound side being unopposed—is largely the true one.

It is probable that a similar train of symptoms might have come on at any time, even as the result of comparatively slight irritation. The serious illness which he had a year before his admission to the Asylum was post-epileptic, and must have been, to some extent, of the same kind as above described.

There is no reason to suppose that the usual vascular changes which take place in the brain as the result of slight or moderately severe shock were absent here. The difficulty is to understand how, in the absence of any apparent inflammatory changes, the paralysis should have persisted, though on the sixth day there was an attempt at recovery, as evidenced by the return to a dreamy sort of consciousness; yet it is to be noted that on that day the loss of motion and sensation on the right side was as marked as before. It is possible, though it is purely a conjecture, that the relapse which occurred next day was due to an unobserved epileptic discharge. Essentially the fault appears to have been a molecular one, and the persistence of the disease was not due to vascular changes so much as to the continued inability of the brain-cells to appropriate such nourishment as was necessary for their activity. In some respects it is allied to cases of functional hemiplegia and hemianæsthesia. Dr. Wilks, in his lectures on 'Diseases of the Nervous System,' says that, in his experience, a perfect hemianæsthesia has been always functional. The anæsthesia of the hypnotic state to which those strange cases of hysterical anæsthesia, of which we have been hearing so much of late, are closely related, presents us with numerous illustrations of temporary loss of function of certain portions of the brain. Such phenomena occur chiefly, if not entirely, in those whose nervous centres are unstable and easily exhausted, and whose power of inhibiting lower centres by higher is very feeble. In such persons any slight and continued irritation, whether it come from the ovary or from the operations of the mesmerist, soon exhaust the centres to which the irritation is propagated, exhaustion rather than increased tension being the condition to which certain groups of cells are reduced. In the light of such considerations and of the imperfect development of this boy's brain, what at first was a very puzzling case becomes comparatively clear.

AN OBSCURE CASE OF BRAIN DISEASE.

BY MONTAGU HANDFIELD JONES, L.R.C.P.

MENTAL derangement extending over six years; absolute deafness; lupoid disease of nose; death from catarrhal pneumonia; autopsy; hæmatoma; syphilitic disease of cerebral arteries.

The following case, which was seen at different times by several distinguished men in London (including Dr. Russell Reynolds, Dr. Sieveking, and Dr. Monro), and the autopsy of which I performed at my father's request, may perhaps be deemed worthy of perusal by the readers of this journal.

Mrs. B., æt. 49, *seen Feb. 6th,* 1874.

On the evening of December 1st, 1873, she was taken with violent giddiness and sickness, this latter symptom persisting for two days; she had not eaten anything that could have disagreed. Since that time she has suffered from "a wavering" in her head, and a weight and tightness at the vertex. She cannot endure music now, though she was an excellent musician. For the last year and a half she has been getting deaf. In 1851 she had a touch of brain fever. Has never had any children. Catamenia ceasing.

At present she is nervous and anxious; complains much of the wavering in her head; is very fidgety about trifles; the deafness is rather increasing; she complains of a sensation of grittiness in the eyes. She needs support in walking, or else she would seem "tipsy." On Feb. 19th she had a species of fit, in which she was unconscious for two or three seconds. Her hands tremble at times, especially the right when she holds it out. Throwing head backward relieves the giddiness. The fundi of her eyes are pale.

About end of March 1874 her condition is the following: No better. Urine pale, sp. gr. 1011, alkaline, not albuminous; she feels some sickness at times; is unable to read, as the letters all run together into up and down lines. She has to keep her feet well apart in walking, and to watch them; when walking she totters much, and seems afraid of falling

backwards. Has difficulty when following my finger to keep both her eyes fixed on the object, as one starts aside, when about 30° from median line. Left pupil slightly larger than right; she often sees double. Grasping power of both hands equal.

On *April 9th*, 1874, some difficulty of swallowing is reported, and fourteen days later this symptom is still present. On the 18th she was suddenly taken with ptosis of left eyelid, and much dimness of vision in both eyes. The left eye is the worst, and if it is partially open she has double and distorted vision; objects on the wall are all V's and A's, *i.e.* the boundary lines are displaced and angular. Her two eyes, as she has often observed, have different foci. She walks about still, but in a very unsteady manner.

April 28th.—Left internal rectus is much paralysed; globes do not move harmoniously. There is almost complete "*loss of speech*" to-day.

May 2nd.—Ptosis much less. Says she can't see, but does really, even if objects are very small. Is unable to work or read. Speaks a good deal, but is often at loss for a word; she uses absurd expressions, as "jenkins" for "pills." Mind seems weak, but she converses with her friends. By the 11th she has lost still more the memory of words, and cannot recall her friends' names. Mourns over her loss of sight, and says that she is going "blad," "blide" meaning to say "blind."

May 18th.—No material change; her morbid state seems to be largely mental, for while she goes on repeating continually "I can't see," and fretting that she cannot read or work, she undoubtedly sees to a considerable extent; this morning she called her husband's notice to two thrushes hopping about in the garden. Her hearing is decidedly improved, there is no ptosis; but the movements of the globes are not quite harmonious. No paralysis of the limbs. At times is violent; has difficulty in making persons understand what she wants.

May 21st.—Improving, but cannot read yet; this seems to be more from difficulty in conceiving the sounds of printed characters than in simply seeing them. She says that she cannot comprehend them, has not the sense, that she is mad. Shows me her collection of post-office stamps, but cannot remember or make out what country each belongs to. Laments that she cannot keep her account books. The defect is evidently in the intellectual centre chiefly; she says she feels strong, she is incessantly chattering, and displays a morbidly increased, though deteriorated, mental activity.

May 25th.—Says she knows everything and can do nothing. She knows what she wants done, but cannot express herself to say what it is. She uses wrong words at times, or words very

partially resembling those which she wishes to use. Gets quite impatient and excited, because she cannot read and work and manage her affairs. Walks well.

May 28th.—She has now had for about a fortnight pretty regularly alternating good and bad days; one day very excitable, petulant, complaining, and troublesome; the next much more cheerful, amenable, and rational. She can certainly see, but cannot read, or understand what is read to her. She certainly hears and walks materially better. She professes to forget names, even her own and her husband's; but it is evident that she can pronounce them with a little effort.

June 1st.—Continues to have good and bad days alternately, but the difference consists only in her being fretful, petulant, and desponding on the bad days and more contented on the good; there is no variation in the inability to read or understand what is read. Sees well, though she says she can't; she went to-day to her bird-cages, and remarked that the claws (very tiny ones) of one bird wanted cutting, but that she could not do it. There is this notable difference between her state and that of another patient I had, viz. that the latter could, though quite an old man, readily understand what was read to him, while Mrs. T. is quite unable to do so, though she can converse to a considerable extent. She says she is quite strong and knows everything, but somehow seems unable to use her knowledge. She missed a piece of work she had netted, and was quite in a temper about it; in enquiring for it she calls it a handkerchief.

June 15th.—Talks a good deal and uses many words, but says she can't talk, and replies to a question put to her, "I don't know what you mean." Shakes her clenched fist with vexation that she can't read. Her brain can evidently take cognizance of objects, and she has the memory to a considerable extent of words; but she cannot use language easily, and especially she has lost the faculty of apprehending the meaning of graphic characters, or of forming them, so that reading and writing are lost to her. She swallows now quite well. Ptosis gone. No paralysis anywhere.

July 2nd.—Seen by Dr. Russell Reynolds, who thinks there is some quasi-inflammatory process at the base of the brain, but no actively growing tumour. ℞ Hyd. Bichlor. gr. j. Pot. Iod. ʒ ij. Aq. ℥ iij. Capiat ʒ ij t. d.

Her state remains unaltered, she is melancholic, says that all food or drink is "blue, green, nasty poison," effervescing lemonade chokes her; she is querulous, morose, miserable. Said the other day, "Is it not a lamentable thing for me to be in this state?" The septum narium is perforated by an ulcer very near the columna nasi (lupus). She seems quite well in

bodily health; eats rather voraciously at night. Very morose and fretful, becomes very passionate at times, and when remonstrated with, says "that she was a lady once, but is not now." Declares she can't talk, can't hear, can't see, but does all three notwithstanding. She complains that she is inside and we are outside, meaning to express how she is cut off from the outside world by reading. She said to me, that I could not think how much she cried inside, meaning that she wept as it were in her mind, though there were no evident tears. She is very active, always fidgeting about, complaining that things are not in proper order; still retains her natural love for animals.

August 17*th*.—State much the same. She continues keenly alive to her loss of reading power, and frets much about it. The right ear is deaf, the left she can hear with. Her repertory of words and expressions is, her husband says, almost perfect, but she has forgotten her music almost as sadly as her reading.

Sept. 17*th*.—Aspect of features degraded as in insanity; same complaint that she cannot see or hear; says that any noise distresses her, goes through her head, yet she bore a short railway journey well. No power of reading or writing. Played cards one day, but complained that she played like a machine. Says she does talk such nonsense. Articulates perfectly, and has throughout.

Nov. 10*th*.—After a visit to Littlehampton she has improved greatly in memory and expression of thought and words; can write now a tolerable letter, but she cannot read yet. She knows however her husband's letters when they come, and is glad to have them, though she cannot read them.

June, 1875.—The lupus has spread slowly and eroded the outer part of the right nostril slightly, the septum is largely perforated. Her memory is good, she can play at cards, plays on the piano a little, but cannot read. Frets much, is decidedly inclined to be violent. Repeats again and again that she used to be a lady, to be so happy and clean, now is dirty, and can't bear to be washed. Says that her brain is broke, and that she can't walk.

March, 1876.—Had an attack three weeks ago, in which for a few days she quite lost her appetite, but had no fever or catarrh; since then she has been *stone deaf* in both ears. Also has become tottery on her legs; cannot walk up and downstairs, as she used. She sees perfectly; reads her name as I write it at the top of my prescription, she knows that latter is for her, says she won't take it. Memory good, eats and sleeps well; is violent and destructive, looks wild and haggard.

December, 1876.—Same condition, is stone deaf. Repeats

the old remarks that she was a lady, now is not: is dirty and hates being washed, throws herself about. No paralysis, walks upstairs in the dark, dresses herself quickly; can see very small things, reads letters, recollects events well and notices objects. Is always complaining of having had her teeth filed years ago. Nose in same state. Pulse not at all cordy.

December, 1877.—Has had an attack of jaundice for several days; mental state much the same; always harping on the old story of having had her teeth filed. Played a game of chess the other day.

January 3rd, 1880.—General health good. She reads now a good deal, reads commonly and understands written signs. Can only be communicated with by writing or signs, as she is stone deaf. She has no paralysis, throws herself about very much; is on her legs all day long. Always fretting about her teeth having been filed; says her nose is gone—all gone, and her teeth and her fingers near the tips. Plays chess well, dislikes being beaten. Is in many respects very rational; but Dr. Monro, who saw her at once, pronounced her insane, and offered to certify her. Says she cannot wash, is obstinate and very refractory, dislikes her nurse. Sleeps well. The sore which used to bleed and be scabbed at the right nostril has cicatrized after destroying a part of the septum and right ala. She dislikes now going outside the house. Is very fond of her husband, can't bear that he should be out of sight, but is more tractable in his absence.

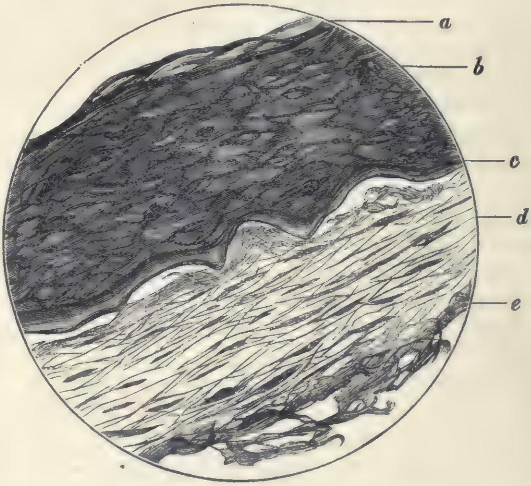
[*Treatment.*]—Iodide of potash and bichloride of mercury had both a fair trial, but without success; and as patient was very refractory the drugs were not forced.

July, 1880.—Died of broncho-pneumonia without any material change having shown itself in her recorded condition.

Post-Mortem of Mrs. B., October 1880. [Weather fine and dry.] Body emaciated; a good deal of hypostasis; scalp pale; a moderate amount of congestion of vessels of dura mater is present.

Dura mater carefully divided along level of sawn bone, and then the brain removed in the usual manner, though still covered by the dura mater.

On examining the surface of the brain, it was found that both hemispheres were covered by "a false membrane," which was adherent to the dura mater externally, and the cerebral arachnoid internally. Anteriorly this membrane extended under the frontal lobes as far as the lesser wings of the sphenoid bone, but in this position it became excessively delicate. Posteriorly it could just be traced to the internal occipital protuberance, while laterally it was just lost below the level of the horizontal limb of the Sylvian fissure. This membrane was



a. Epithelioid Lining.
b. New Growth.
c. Membrane of Henle.
d. Muscular Coat.
e. External Coat.

To face p. 547.

of a buff colour, homogeneous throughout in structure, firm and tough, showing on its internal surface at the parts where it was the thickest, peculiar stellate groups of injected vessels; the largest patch of these vessels was about the size of a halfpenny. By its outer surface the membrane was closely adherent to the dura mater, while between its inner surface and the cerebral arachnoid numerous well-marked vessels and what appeared to be fibrous bands were seen to be passing. The thickness of the membrane varied considerably—thus in the longitudinal median fissure, on the surface of the horizontal plate of the frontal bone, and laterally below the horizontal limb of the fissure of Sylvius it was a mere film; posteriorly over the occipital lobes, the angular, and the back part of the supra-marginal and superior parietal lobules, it was rather thicker than ordinary writing-paper; while at its thickest part over the three superior frontal convolutions, the ascending frontal and ascending parietal, and front part of the superior parietal and supra-marginal lobules it was fully an eighth of an inch in thickness.

The dura mater lining the cranial bones appeared natural; the vascular grooves on the bones were well marked. There was no thickening of the arachnoid. Along the margins of the longitudinal sinus (superior) small nodules of Pacchionian glands were very apparent.

The base of the brain, with the cerebellum, pons varolii and medulla oblongata, appeared normal. The arteries were large, gaping, and thick-walled; but there was no appearance of embolism in any of them.

On removing the false membrane the hemispheres were seen to be quite natural; the convolutions were not unduly flattened, nor abnormally adherent to each other. The auditory, facial, and other cranial nerves were quite natural: the two first-named lay normally in the meatus. On cutting up the brain the veins of the lateral ventricles were found rather large and full, but no tumour, undue softening, or other signs of morbid action could be traced in any part.

Microscopical Examination.—Sections from the hæmatoma showed that the latter presented all the features of a blood clot, which was becoming gradually organised. The vessels of the pia mater appeared to be perfectly healthy. Sections of the grey matter taken in varying situations were found quite normal. Examination of the large arteries at the base of the brain showed that important changes had taken place in these vessels. These changes which are represented in the adjoining sketch show clearly that the morbid process had its seat in the inner coat of the artery only; there was in fact a great proliferation and overgrowth of the connective tissue cells which

are normally found between the epithelioid lining and the fenestrated membrane of Henle. This chronic endarteritis has been fully described by Heubner in his admirable monograph on disease of the arteries of the brain, and is doubtless of syphilitic origin. The thickening of the coat of the artery varied at different parts of the wall; thus, while on one side of a section a considerable growth of connective tissue was apparent, at the opposite side frequently it could be made out that cell proliferation was only just commencing. In addition to this fibroid change it was clear that the disease tended to injure and impair the structure of the artery; thus, in many places the epithelioid lining was rough and ragged and projecting, at others the muscular layer appeared thinned and degenerated, while in other parts there seemed to be a disturbance of the connection and relation of the different coats one to the other.

In reading over the case reported above, two points seem brought prominently before us; firstly, that we have here an undoubted case of insanity, and secondly, that we are able to point to a well-marked and definite lesion as to the cause of the disease.

That insanity was present can scarcely be doubted, especially when such an eminent physician as Dr. Monro is willing to certify the case as such; and yet in some points the symptoms do not fully bear out such a statement. Thus the patient was undoubtedly at times melancholic and desponding, yet we never find that she regarded those around her with aversion or hate, as is so often the case; on the contrary, she retained her intense love for her husband and also her partiality to her pets quite unaltered. Apathy and desire for solitude are also frequently marked symptoms in cases of melancholia, but Mrs. B. always displayed considerable interest in her household matters, and never ceased lamenting that her infirmity prevented her from giving the needful care to these subjects. As regards solitude, moreover, the patient, though naturally confined almost entirely to her home, was yet always rambling over her own house, and seemed glad of the society of visitors when they came to call on her. Again, her consciousness of her own defects is painfully apparent. It is not that she has a morbid idea of a state which exists only in her own fancy; but she is dirty in her person, she is unable to attend to her house, she is disabled from music, reading, &c., and it is under a sense of these "real failings" that she suffers so acutely. Undoubtedly at times she was violent and refractory, but if we examine her actions at these times we fail to trace anything akin to maliciousness or distorted ideas; rather, the acts are those of a brain which is

irritated and upset from its true balance. Her own expression, so constantly used on these occasions, viz., "I can't help it," seems to point to the fact that the right sense was present, though some disturbing influence produced ever and anon an error or misdirection of cerebration. It seems important that we should recognise these points, because later on we shall find that these manifestations of disease are quite such as we should expect from the lesion present.

When we turn to the evidence afforded by the post-mortem examination, what we find is shortly this:—A large hæmatoma spread out symmetrically over both hemispheres, continuously but loosely adherent to the dura mater externally, more firmly but quite as universally attached to the arachnoid and pia mater below; in other words, over the grey matter of the convolutions, *i.e.*, over the substance which is without doubt the seat of the higher intellectual faculties, and probably in certain areas forms the centres for special mechanisms, we find superimposed a large irritating foreign body.

The symptoms of hæmatoma, as given by Aitken, Schuberg, and others, consist principally in general weakening of memory and intelligence, in giddiness and local head pains, in transitory fits of unconsciousness, in impairment of the power of speech, in excessive appetite, and lastly in paralysis of the face or limbs, the duration of this latter symptom varying greatly in different cases. While, however, we find that certain symptoms, as giddiness and weakness of intellect, are invariably present, it is evident, with regard to other symptoms, that many modifications or complications will arise, according as the extent, relations, and connections of the tumour vary. Regarding, then, in the present case the nature of the lesion, we can without much difficulty picture the character of the lesions likely to arise. Thus motor disorders will probably be present, and in the report we do notice that tottery walk, want of harmony in the action of the two eyes, ptosis of the left eyelid, and paralysis of the left internal rectus, were prominent symptoms. It is important to note that these paralyses came early in the disease and soon disappeared; for, as Goltz has shown in dealing with injuries to the convolutions, such temporary phenomena are due to the superficial lesions exercising inhibitory influences on the parts of the brain lying between the convolutions and the spinal cord, and not to any organic injury to these parts themselves. Again, since the false membrane was thickest and most adherent over those portions of the cerebral convolutions which are usually marked out as the centres for speech and sight, and therefore the irritation to these parts was likely to be considerable, we should expect to find that dis-

orders of speech and sight played a prominent part in the history of the disease; and such was actually the case. That mental disease should have shown itself markedly, is only what must have occurred under the circumstances. Dr. Wigan, in his work 'On the Duality of the Mind,' quotes a case in which a very slight depression of bone, caused by a tap on the head with a ruler, led to a complete change in the moral feelings of the patient; and if such a result could follow so slight a source of irritation, what wonder if the state of mind with which we are here concerned should have so prominently manifested itself! Moreover, in Dr. Wigan's case there was but one focus of irritation, and that limited; while in Mrs. B.'s case the whole mass of the convolutions was exposed to the fretting and irritation which must of necessity have arisen from their contact and connexion with the hæmatoma. The form too under which the mental derangement showed itself was striking. It is usual, in cases of hæmorrhage into the cavity of the arachnoid, to have intellectual disorders; but these usually appear in the shape of dulness and stupidity, somnolence and apathy, while in the present case quite the opposite state existed. There was, in fact, great mental activity, but it was of a morbid and deteriorated quality; the quick, bright habit of cerebration natural to the patient was not lost, but the brain was working under a cloud, was, in fact, working under some abnormal influence which misled and overpowered its normal healthy action. Her own complaint "that she was inside, and others outside," exactly hit off the state of the case; for between her own inner consciousness and her natural healthy relations with the external world disease had erected an impassable barrier—a barrier the existence of which she fully realised, but the influence and restraint of which she could not in any sense control. Regarding the deafness, which was so marked a symptom in the case, it is noteworthy that it had existed in a less degree before the patient's illness commenced, and that there is not sufficient evidence to connect it in any way with the intracranial lesion which was found. The case is of interest as showing that some forms of insanity can certainly be attributed to intracranial lesions, while at the same time it points to the fact that adverse conditions, which produce only a comparatively slight and temporary derangement of motor mechanisms, are quite sufficient to cause extensive and lasting injuries to the higher centres of intellect and volition.

While thus far we have attributed all the morbid symptoms to the existence of a large hæmatoma, we must not forget that the presence of lesion in the arteries of the brain may have played an important part in the causation of the disease. In

reading over Heubner's researches on syphilitic disease of the brain arteries, two points can scarcely fail to strike us, viz., firstly, that the morbid state of the vessels in the case under consideration is in reality the same as that which he describes and pictures; and secondly, that many of the symptoms which he considers diagnostic of syphilitic disease of the brain arteries were undoubtedly present in Mrs. B.'s case. Two extracts from his paper will illustrate these points. At page 224 he writes thus:—"The second principal group of symptoms of our disease next to those of hemiplegia (or the third group, if we look on the short-lasting attacks of unconsciousness which introduce the hemiplegias or come on without such, as the first) turns on the injury of the higher intellectual functions, on the damage of consciousness, and thus on the functions of the grey matter of the brain. The disturbances relative to this latter are not less frequent than the paralyses; indeed, we may even say that they exist more constantly; moreover, they are not absent at times when paralysis shows itself only in a significant manner." Again, at page 225 we find, "The characteristic of this universal brain disturbance is, in a word, the incompleteness, the imperfection, of the severe symptoms, *e.g.*, the injury of the consciousness without its complete removal, the giving up of absolute activity without removal of the impulse of the will, the appearance of delusions and delirium, but the sudden return at times to reason; it is a state of partial somnolence, partial wakefulness, partial dreaminess." Against the idea, however, that arterial disease played a prominent part in producing our patient's troubles must be brought the fact that no softening or other morbid change in the brain substance, such as we should expect if the blood supply of the nervous tissue was seriously interfered with, could be found at the post-mortem; on the contrary, there was every appearance of the cerebral circulation having been as full and active as it normally is. That disease of the cerebral vessels may have caused the hæmatoma, or even may have aided in the development of the symptoms present, is quite possible; but to imagine that the amount of arterial disease existing was sufficient *per se* to produce any serious deterioration of the cerebral functions seems scarcely borne out by the evidence before us.

Abstracts of British and Foreign Journals.

Magnan on Word Blindness. (*Gazette des Hôpitaux*, January 24, 1880.)—M. Magnan, in a communication to the Société de Biologie, relates two cases of aphasia complicated with a special phenomenon, to which he has given the above name.

One case was that of a man who was seized with right hemiplegia and aphasia after a fall. A month afterwards the patient recovered the power of speech, little by little; he understood spoken language; he wrote, either of his own accord or from dictation; but he was incapable of reading either print or manuscript, even when the latter had been written by himself; and he could not name letters inscribed upon a board.

The second patient presented similar symptoms. He recognised objects which were shown to him, but could not name them; could write words thought or heard, but could not comprehend or copy what was written. He had lost the notion of the value of gesticulations.

M. Magnan also cited a similar case, reported by M. Brouardel, where on post-mortem examination a hæmorrhagic centre was found at the posterior part of the fissure of Sylvius adjoining the pli courbé.

He explains the pathology as follows. The retinal image is impressed on a first centre, at the level of the quadrigeminal tubercles; that is, the reflex centre for the eye. From this centre it is impressed upon a second, situated about the pli courbé, or perhaps about the occipital convolutions. In the second centre it is perceived as a sensation and received by the attention and the memory; this being the psychic visual centre. The ideas to which its elaborations in this centre have given birth cannot be utilised for speech, unless the communications between it and the convolution of Broca are intact. If they are interrupted, the patient can still see, speak, and hear, but he cannot acquire through his eyes any new idea.

As there is no disease of the eye, and because it is owing to a purely psychic phenomenon, this affection might be better described as "Cerebral Word Blindness."

Delaunay on Memory.—M. Delaunay has made a communication on the memory to the Société de Biologie, the result of extended inquiries. His results are classed under the following heads:—

Species.—Memory is a prominent faculty among birds and among certain mammals—the horse, elephant, &c. For example, a horse often has a better recollection of routes and localities than his master.

Race.—Memory was very highly developed among ancient nations, evidenced by their long poems, handed down traditionally before the invention of writing. The inferior races have better memories than the superior—the Negroes, Chinese, Italians and Russians who learn foreign languages with extraordinary facility.

Sex.—The adult woman has a better memory than a man. Actresses learn their parts more quickly than actors.

Age.—Young people have better memory than old. The faculty attains its highest development at thirty years of age, and thereafter declines.

Constitution.—The feeble are more highly endowed with memory than the robust; provincials than Parisian, country-folk than townspeople, lawyers than medical men, ecclesiastics than the laity, musicians than other artists. At the normal schools, Val de Grâce, &c., the pupils who have the best memory are not the most intelligent.

Individual Organs.—The right cerebral hemisphere has more of the faculty of memory than the left. Lesions of the former affect the memory more than the other faculties; lesions of the latter leave memory intact. It is common to all the nervous system. The organs which preside over walking, swimming and flying are endowed with more stable memory than those of the senses; those of the senses than those of the emotions; those of the emotions than those of the intellect.

Physiology.—Memory is more active before than after a meal. Cerebral congestion interferes with this faculty. Education diminishes it, and the illiterate man has a better memory than he who can write. Morning is more favourable to memory than the evening; summer than winter; the south than the north.

Pathology.—The memory is in a state of exaltation in certain maladies, such as hysteria, and depressed by poisons, typhoid fever, &c.

In conclusion, memory being the gift of inferior races, women, adolescents, the feeble, the less intelligent, the right cerebral hemisphere is in*inverse ratio to the evolution.* Still more so it is in a certain measure in *inverse ratio to the nutrition*, because it is augmented by circumstances which diminish the nutrition.

M. Delaunay proposes to apply the same method to the study of all the faculties. He divides them into two great groups. The first, the inferior, including imitation, superstition, &c., attain their maximum in the case of inferior beings—women, children, old men, feeble and in the right brain. The second, the superior, including conception, comparison, invention, &c., are best seen in the superior races, white races, masculine sex, the adult, the strong and the left brain.

A. R. URQUHART, M.D.

Buzzard on Transfer-Phenomena in Epilepsy produced by Encircling Blisters.—In the course of this paper, which was read in the Medical Section of the Meeting of the British Medical Association at Cambridge during the past autumn, the author referred to four cases published by him in the 'Practitioner,' October, 1868, in which remarkable results followed the application of blisters (especially encircling blisters) to "limbs" which were the seat of marked epileptic aura. In one, a tickling in the left arm had always preceded the fit. After the application of a blister encircling this limb the tickling was transferred to the left leg. In another, characterised by a similar aura, the fits and the tickling ceased after the application of an encircling blister. In a third, a sense of numbness in the left wrist was transferred to the right wrist. A fourth was a woman in whom fits had always been preceded by cramp in the right hand, and who after the blister was affected with cramp in both hands before her fits. In one of these cases a subsequent autopsy showed cerebral tumour. Dr. Buzzard had recently applied encircling blisters to some other cases with the following results. In one where the fits (two years' duration) had always been preceded by cramp in the left foot and shaking and numbness in the left leg, after the application of an encircling blister to this limb it was the *right* leg which shook and was numbed. In another, tingling in the left arm was the symptom, and after blistering there was jerking of both arms and the left leg. In a third, the attacks were preceded by cramp of the left hand. The patient had had a severe fit two days before being seen. The left hand was quite powerless. An encircling blister

to the left arm was applied, and a week after the patient complained that though her left hand had next day regained its power the right hand had *ipso facto* become weak. The dynamometer showed a power of grasp of 40 k. with the left hand and only 18 with the right. The author's original observations had been made many years before those experiments upon hysterical hemi-anæsthesia in France, in which removal of the anæsthesia was found to be accompanied by its transfer to the other side. He urged that the phenomena pointed to a power of influencing in some way nervous centres by impressions upon the skin, and referred to some trials of a therapeutic character which he was basing upon the observations. His aim was in a case of aphasia to rouse into activity the posterior portion of the third frontal convolution of the *right* hemisphere by directing powerful impressions to adjacent grey matter by means of painful electrical currents applied to the tongue, mouth, and left arm.

J. Hughlings-Jackson.

Luederitz: Experiments on the Effect of Pressure on Motor and Sensory Nerves. (*Zeitschrift für Klinische Medicin*, Bd. ii., 1.)—In the introduction of his interesting paper, the author observes that a considerable number of cases have been reported in which peripherical mixed nerves were entirely cut in two without producing loss of sensation in the corresponding parts, the paralysis of the motor fibres of the nerve being at the same time complete. *Richet* noticed in a case of neurotomy of the median nerve, that when he happened to touch the peripherical end of the nerve the patient gave signs of violent pain. Evidently in this individual that part of the median nerve contained sensory fibres which were in connection with some other brachial nerves. A similar case of this "sensibilité récurrente" is reported by *Remak;* afterwards *Arloing* and *Tripier* proved by experiments on animals that many nerves contain sensory fibres derived from sensory nerves in the neighbourhood; these fibres run a little way in a centripetal direction, and then enter into the nerve to which they really belong.

These facts explain to a certain extent those well-known clinical cases in which we find complete motor paralysis without loss of sensation; but there are other cases which cannot be explained in this way, and therefore the author made a series of experiments on rabbits in order to ascertain whether the resistance to injury of sensory nerves is greater than that of motor nerves. He applied a

ligature to the sciatic nerve, and then stimulated with a faradic apparatus both the central and the peripherical part of the nerves at a few centimetres distance from the place where the ligature lay. The distance of the two coils, divided in millimètres, expressed exactly the intensity of the current which was wanted for the production of a contraction of corresponding muscles, viz. of a sign of pain in the animal. In order to avoid the influence of "sensibilité récurrente" the crural nerves as well as all other sensory nerves of the leg were cut so that there was no sensory communication between the extremity and the brain but the sciatic nerve.

The result of these experiments was as follows:—The conductivity of motor fibres is invariably impaired earlier than that of the corresponding sensory fibres. It was found several times that 10–15 minutes after the application of the ligature sensibility and motility disappeared simultaneously, but much more frequently the author stated that the strongest faradic current applied above the ligature did not produce the slightest muscular contraction, whereas peripherical stimulation immediately afterwards made the animal display symptoms of pain. Sometimes motor conduction was completely interrupted, sensibility being at the same time absolutely unaltered. On the other hand, loss of sensation without motor paralysis was not observed in a single case. When the ligature was removed in cases of complete motor and sensory paralysis, in some cases both faculties were restored at the same time; in other experiments sensation appeared to have returned, while motor paralysis persisted.

The only way to explain these facts is to assume that sensory nerves have a greater resistance against traumatic influences than the motor nerve-fibres. As a practical consequence, the author points to Duchenne's observation: that the prognosis of traumatic paralysis is much more favourable when, the electro-muscular contractility being extinct, muscular sensibility is unaltered or but slightly diminished.

Brieger: A Case of Paralysis from Fright. (*Zeitschrift für Klinische Medicin*, Bd. ii., 1.)—The popular idea that violent mental shocks are capable of producing severe illness has not gained much ground in scientific researches. Only lately some cases have been reported by Kohts and Leyden, giving indubitable evidence of disease caused by intense fright (during the siege of Strasburg in the late Franco-German war). Similar ob-

servations have been made by Todd, Hine, Pasque, Berger, but all these were deficient with regard to anatomical examination. Leyden only has made autopsies in two cases of spinal paralysis: in the first case he found sclerosis of the middle part of the dorsal region of the cord; in the second case there was diffuse sclerosis of the upper and middle part of the cord in the dorsal region. The following observation, made at Professor Frerich's clinique, will therefore attract the attention of pathologists.

A prostitute girl, 23 years of age, without hereditary disposition to nervous diseases, going home late at night from a dance, ran against a man who was lying drunk on the stairs. She managed, however, not to fall, by clinging to the handle of a door, and there was certainly no definite injury to the spine. Trembling with terror she mounted with difficulty to her room in the third story and tumbled on her bed, almost fainting. After a little while she recovered from her fright, undressed and went quietly to bed, without feeling anything abnormal. At 4 o'clock in the morning she awoke, in consequence of intense pressure of urine, but found herself incapable of passing any water. At the same time she felt violent shivering. Hot bottles and tea applied by the landlady did her no good; she therefore went into the warm bed of the latter, in doing which the patient observed a considerable weakness in her legs. She then went to sleep; when she woke at 7 A.M. she found her legs entirely paralysed, and at the same time had a sensation of numbness and tingling. A few days afterwards she was brought to the hospital. There was now complete paraplegia, pain in the lumbar region when she tried to sit up, sensation of cold and heaviness in both legs. Mental functions normal, great depression of spirits. No fever. Residues of syphilis, for which she had been treated at the same hospital two years before this accident. Absolute anæsthesia of the lower part of the body up to the height of the second lumbar vertebra. Retentio urinæ. The treatment consisted in application of 4 grammes of mercury ointment pro die, and 2 grammes of iodide of potassium internally. No improvement in the following days. On the 12th day sloughing began in the sacral region, extending rapidly. A few days later, symptoms of spinal meningitis came on. Patient died on the 27th day after her reception at the hospital.

The *autopsy* revealed diffuse myelitis, extending from the upper lumbar region to the level of the 8th dorsal vertebra, chiefly localised in the posterior columns and the parts of the white

substance adjoining the anterior roots. Upwards there was found secondary degeneration of the postero-median columns (Goll's columns) reaching as far as the fourth ventricle of the brain.

The author thinks that in this case there can be no doubt as to the cause of the disease; he does not believe that the syphilitic history of the patient has anything to do with it, because there were no symptoms of visceral syphilis, and the other residues of the infection had been quickly improved by the anti-syphilitic treatment. He only admits that the patient's resistance against disease had been diminished by the venereal infection. Concerning the way in which mental shocks act on the cord, the author suggests that the fright brought on a sudden intense contraction of the blood-vessels, lasting for a considerable time and leading to necrotic destruction.

R. H. PIERSON (Dresden).

Onimus on Modifications in the Excitability of Nerves and Muscles after Death. (*Journal de l'Anatomie et de la Physiologie*, 1880, No. 6.)—We know that after death muscles and nerves gradually lose their excitability. The vital functions are rapidly, almost instantaneously, lost as regards the brain, while in the spinal cord they persist for a short period, the grey matter losing its experimental excitability sooner than the white matter. With regard to the nerves, the loss of excitability does not, strictly speaking, occur in a regular order, commencing from central parts and gradually invading peripheral parts; the loss of excitability pervades the nerves of a limb as follows: first, the large cords; secondly, the filaments supplying extensor muscles; thirdly, the filaments supplying flexor muscles—a sequence that calls to mind common pathological events, e. g. dropped wrist. The author states that in the arm the radial nerve, in the leg the peroneal nerves are the first to lose their excitability.

The nerves of the ganglionic system preserve their excitability longest of all; the intestine has been seen to contract five or six hours after decapitation, and M. Robin elicited rhythmic pulsation of the right auricle many hours after death by distension of its walls. With regard to the muscles, their excitability during the first few minutes after death is enhanced; they are at their maximum excitability when the large trunks are inexcitable, their excitability is already on the decline when the smaller filaments are inexcitable. After a gradual death, which is an exhausting process, muscular farado-contractility is more rapidly lost than

after a sudden death; the decline is more rapid in birds than in mammals, least rapid in amphibians. Analogously with the event as regards nerves, extensor muscles lose their farado-contractility before flexor muscles (an hour or more). There is no appreciable difference between muscles of the superior and muscles of the inferior extremity. Muscles of the trunk and abdomen are the last to lose their farado-contractility. The author remarks that just as nerves which are most easily influenced by lesions during life, are the first to lose their excitability after death, so in muscles we find a similar correspondence; facial muscles going first, extensor muscles going before flexor muscles, muscles of the trunk going last. With regard to the efficacy of various stimuli, the greater energy of response to galvanism with diminished or absent response to faradism, commonly noted in pathological cases, is not observed as a post-mortem phenomenon, although very feeble galvanic shocks are competent when the strongest faradisation is ineffectual; also the contraction is tonic, persists during the passage of the current, and is not merely a spasm at its make and break. The above statements apply of course to percutaneous stimulation—we know in effect, that when with unbroken skin a muscle does not respond to faradism, it will still respond if the stimulus be applied to its bared surface; but the contraction now elicited is local, not general, and the author remarks that probably the stimulus does not act quâ electrical, but quâ mechanical, for at this period mechanical irritants are highly efficacious.

The approximate chronology of the events as regards muscle is as follows:—

$2\frac{1}{2}$ to 3 hrs. after death,		the muscles of the face and tongue, with the exception of the maseter,	have lost their farado-contractility.
3 to 4	„	the extensor muscles of the leg and arm, and the maseter	„
4 to 5	„	the flexor muscles of the leg and arm	„
5 to 6	„	the muscles of the trunk	„

With regard to the decline in nerves, the author states that of the large trunks excitability is lost at the end of 2 hours, that of the small branches flexor filaments retain their excitability often 4 hours longer than "the others" (extensor filaments? large trunks?).

The chronology of muscular galvano-contractility is not exactly laid down, the only statement on the point being that as faradism

loses efficacy, galvanism has a more marked action, and at a variable time after death remains alone efficacious; mechanical stimuli acquire a relatively greater action than electrical stimuli, and are finally the only stimuli capable of eliciting contraction; the last electro-muscular reaction which the author obtained was 9 hours after decapitation, being a local, sluggish, prolonged contraction to the direct application of the galvanic electrodes, apparently a result of chemical action.

As a sign of death, the author concurs in Rosenthal's opinion that electrical exploration is the best, most certain, and most rapid test of that event; the approximate time after death may even be pronounced on the above data. Thus, for instance, if in a limb faradism elicits response from the flexor muscles, no response from the extensor muscles, it may be affirmed that death has happened between 4 and 5 hours previously. If at the same time on the face we more easily elicit contraction by galvanism than by faradism, and if the contraction is sluggish, we are further assured that death has happened about 4 hours previously.

The electrical test fails only where it is necessary to know *rapidly* whether death has taken place, but the only case in which this necessity can arise is sudden death during pregnancy, where there is a possibility of saving a life by the Cæsarian section. In this single instance, other tests need be relied on, namely, the contrast between the appearance before and after sudden death, and the comparison of the maternal and fœtal pulse.

In sum, the electrical test affords the best sign of death, may inform us of the period of its occurrence, and is a powerful means of reawakening the circulatory and respiratory functions.

A. Waller.

Strümpell on the Pathology of the Spinal Cord.—In this paper (*Arch. f. Psych.* x. p. 676, and xi. p. 27), the author discusses the subjects of spastic spinal paralysis and combined systematic disease.

Spastic Spinal Paralysis.—The first case reported is that of a man, aged 25, who had contracted syphilis four years previously. Death happened within nine months of the appearance of the first symptoms. During the greater part of the time the symptoms were those characteristic of spastic spinal paralysis: there was gradually advancing paresis and paralysis of the inferior extremities, excess of the tendon reflexes, rigidity of the muscles, the usual spastic phenomena, and an absence of all sensory symptoms. It was not

however a typical case of the disease, for at an early period transient irritability of the bladder was noticed, and two or three weeks before death paralysis of the bladder and bedsore appeared, sensibility became affected, the muscles of the limbs atrophied, and the tonic contraction of the extensors was succeeded by contraction of the flexors.

The post-mortem revealed the presence of a diffuse chronic myelitis of the upper dorsal part of the cord. Westphal, it will be remembered, has shown that this part of the cord is very frequently affected in chronic myelitis. Though the disease was diffuse, it was observed that certain parts were more prone to be attacked than others, and that a striking symmetry obtained between the lesions on the two sides. Thus in the posterior columns, the columns of Goll were always diseased, and if the lesion spread to the adjoining columns of Burdach it was always the posterior parts that were most affected. In the lateral columns the posterior regions were more affected than the anterior, and the lateral limiting layers were almost intact. The anterior columns showed few signs of disease beyond a thin zone of marginal degeneration on each side. The changes in the grey substance were most marked in the anterior cornua; the most noteworthy appearances being the paucity of the ganglion-cells, the number of large spindle cells, the presence of cells with fatty granules, and, most important of all, the widely dilated vessels which gave quite a cavernous look to the tissues. In other places the adventitia of the vessels was much thickened. The myelitis had a further peculiarity; it did not consist of one large focus, but had a floccular or disseminated character, and presented a strong resemblance to the disseminated cerebro-spinal sclerosis. Indeed cases have been described in which the distinction was so puzzling that the diagnosis of chronic myelitis or disseminated sclerosis rested on the presence or absence of secondary degeneration.

In the latter disease secondary degeneration is not met with above and below the focus of myelitis; tracts of ascending and descending degeneration were respectively observed. There was ascending degeneration of Goll's columns and of the direct cerebellar tracts. The degenerated cerebellar tracts were traced upwards to the medulla. They lay nearer the olivary body than Flechsig figures them. There was descending degeneration of the lateral pyramidal tracts, of the direct cerebellar tracts, and for a short distance of a thin band of fibres on the outer side of each Goll's tract (bandelettes externes). The degeneration of the cerebellar tracts was found

as low as the upper part of the lumbar region. Clarke's columns were, throughout their length, considerably poorer in cells than usual. The connection between these cells and the cerebellar tracts seems much closer than that between the cells of the anterior cornua and the pyramidal tracts, for while disease of Clarke's cells very frequently accompanies degeneration of the cerebellar tracts, we often have disease of the pyramidal tracts without alteration of the cornual cells. How shall we explain this descending degeneration of the cerebellar tracts? Three hypotheses seem open to us. The morbid process may have spread from the pyramidal strands to the contiguous cerebellar tracts, or the case may have been one of systematic disease of the cerebellar tracts complicated with dorsal myelitis, or lastly, it may be that focal lesions of the cord in certain circumstances are associated with descending as well as ascending degeneration of the cerebellar tracts. The last view is adopted by Strümpell.

The slight implication of sensibility is remarkable, considering the extent of the myelitis. As the lateral limiting layers were intact, the author suggests that perhaps they are the conductors of the sensory impressions.

The next case was, as regards its symptomatology, a typical case of spastic spinal disease. The most remarkable abnormality was the excessive dilatation of the central canal of the cord. The dilatation commenced between the origin of the first and second cervical nerves and extended throughout the cord. It was greatest in the dorsal and upper cervical regions. In some places the cavity measured 8 mm. from side to side. In the upper part of the dorsal cord the central canal appeared double on transverse section, owing to a small diverticulum from the canal at a lower level. The dilatation was not the result of softening or shrinking in the surrounding tissue, but was a primary change due to abnormality of development.

Besides the hydromyelus there was degeneration of the pyramidal strands of the lateral columns. The appearances were exactly similar to those found in secondary descending degeneration, but as there was no evidence of direct interference with the tracts, and as the lateral limiting layers, which pressure radiating from the central canal would naturally affect most, were almost intact, as too the degeneration was not confined to the pyramidal tracts, but implicated also the cerebellar strands, Strümpell inclines to think that the case was one of hydromyelus accompanied by primary systematic disease of the pyramidal and cerebellar

tracts. A narrow belt of degeneration, probably the result of an extension of the disease from the central canal, surrounded the grey matter in the anterior and lateral columns. In the posterior columns there was no sign of systematic disease; on the contrary, the affection had an exquisite disseminated character.

Taking a general view of the case, the author remarks that there was a hereditary predisposition to nervous disease, which showed itself in the earlier stages of development in the anomalous condition of the central canal. Probably the faulty laying down of the strands of the cord, some systems being more affected than others, predisposed to the early appearance of the systematic affection.

Spastic spinal paralysis occurs in widely different diseases. We have considered at length two conditions in which it was seen, and to these Strümpell adds, giving cases, injuries of the spine (myelitis from compression), tumours of the cord, multiple cerebrospinal sclerosis, chronic hydrocephalus, and those affections of the nervous system which are sometimes found as the sequelæ of acute illnesses, and especially of typhoid fever. It is impossible as yet to specify the anatomical lesion that underlies the disease, though, as Erb stated, primary systematic degeneration of the lateral columns often induces it.

In pursuing this investigation we must be careful to distinguish between the paralysis and the spastic phenomena. We may have the latter without a trace of the former. We must also remember the complexity of the group of spastic symptoms. This includes, besides the phenomena dependent on excess of the tendon reflexes, various kinds of muscular contraction and contracture, e. g., the contracture due to direct motor irritation (flexion of the leg with convulsive dorsal flexion of the foot, *Beugecontractur*), and the contracture due to reflex influences (extension of the leg with strong plantar flexion of the foot, *Streckcontractur*); and to these must be added localised convulsive movements that are produced by a variety of causes.

Combined Systematic Diseases of the Cord.—The first case presented the group of symptoms found in amyotrophic lateral sclerosis: paresis of the extremities, trophic changes, and spastic symptoms. There was simple atrophy of the muscles of the forearm and hand, but in the triceps and the muscles of the lower extremities, there was, in addition, a striking degree of fatty infiltration of the muscles: the reaction of degeneration was not observed. The most constant of the spastic phenomena was the

strong dorsal flexion of the foot and toes, with which was often combined flexure of the thigh and leg, making up what the Germans call a *Beugecontractur*. The tendon reflexes were excessive. Convulsive flexing movements were observed in the legs from time to time, and generally affected them alternately. The passive contractures observed in paralysed limbs must be distinguished from the spastic contractures just mentioned. The former are permanent, and the deformity of the limbs gradually grows worse. The latter vary in intensity at different times, partly owing to the varying intensity of the central irritation, partly to the varying irritability of the nerves and muscles. In the former there is club-foot; in the latter, dorsal flexion of the foot. Strümpell thinks that the spastic contracture is due to primary disease of the pyramidal lateral columns, the fibres being still in connection with their trophic centres, and hence retaining their irritability. Applying the same reasoning, secondary degeneration of the lateral columns cannot account for the contracture of hemiplegia, for the fibres are separated from their trophic centres, and speedily lose their irritability.

A peculiar feature of the case was the association of movements in the lower limbs. There was not only an abnormal irradiation of the voluntary impulse to other muscles on the same side, but the patient could never flex his thigh either voluntarily or as the result of a reflex excitation, without inducing very intense dorsal flexion of both feet. There was complete incontinence of urine, and ultimately purulent cysto-pyelitis. Sensibility to cutaneous stimuli was perfect. A feeling of cold and formication was experienced in the lower limbs, but never to any great degree. Towards the end, pains of some severity were felt, but they were quite different from lightning pains.

The case was diagnosed as amyotrophic lateral sclerosis, though there were evidently important points of difference between it and Charcot's cases of this disease. For example, in Charcot's cases the disease generally commenced in the upper extremities, and travelled downwards; in this case an exactly opposite course was pursued. Further, in Charcot's cases there was usually atrophy, without fatty infiltration, of all the muscles of both extremities, and there was no paralysis of the bladder. Nevertheless, there seemed good ground to expect systematic disease of the pyramidal strands, and lesion of the anterior cornua.

The lesions found on *post-mortem* examination were (1) complete degeneration of the lateral pyramidal tracts of both sides in their

whole length, and of the anterior pyramidal tract of the right side down to the lower dorsal region; (2) degeneration less intense, of the cerebellar tracts; and (3) degeneration of the posterior columns. The anterior cornua were quite healthy. The cells of Clarke's columns were abnormally few in number in the lower dorsal region. The disease nowhere extended into the medulla oblongata.

The distribution of the disease in the posterior columns was remarkable. The condition of each column was as follows: (1) a narrow belt of normal tissue bordering the posterior fissure; (2) external to this, the degenerated Goll's column, triangular in shape; (3) external to this again, the degenerated 'postero-external region of the posterior column,' also triangular in shape; (4) in the anterior part of the column, and with its apex directed back between the apices of (2) and (3), a third triangular area, of normal tissue; (5) in the mesial line immediately behind the posterior commissure, and at the apex of Goll's column, a small band of degeneration; and lastly (6) a narrow intact strip on the inner side of the posterior roots. The symmetry of the disease on the two sides, and the occurrence of the same sharply defined areas in widely separated parts of the cord, are strong arguments in favour of the systematicness of the affection. (1), (2), and (5) together, make up what is usually regarded as Goll's column. (3), (4) and (6), constitute Burdach's column. The somewhat indefinite 'bandelettes' of Charcot probably correspond to (4).

Viewing the lesions in connection with the symptoms, this case seems to teach us (a) that sensory conduction does not take place either in the pyramidal strands, the cerebellar tracts, Goll's columns, or in the postero-external divisions of the posterior columns; (b) that with very extensive degeneration of the pyramidal tracts we may only have symptoms of paresis; and (c) that the posterior columns may be diseased in the lumbar region, and yet there be excessive tendon reflex.

In a second case, the symptoms were paralysis and contracture of the lower extremities, heightened tendon reflex, incontinence of urine, and a degree of analgesia in the legs. The autopsy gave degeneration of the lateral pyramidal tracts (particularly in the inferior dorsal and lumbar regions), the cerebellar tracts (degeneration most marked here), Goll's columns, and the postero-external divisions of the posterior columns. The anterior cornua were normal. The degenerated Goll's columns were traced to the upper part of the lumbar region.

Allusion is made to a third case, reported by Kahler and Pick,

in which the symptoms bore a strong resemblance to those observed in Friedreich's cases of hereditary ataxia, and where after death there was found systematic degeneration of the pyramidal strands, the cerebellar tracts, and Goll's columns. These three cases, Strümpell thinks, leave no doubt as to the existence of primary combined systematic diseases. The morbid process in these diseases is not a chronic inflammation originating in the connective tissue or blood-vessels, but is a true parenchymatous degeneration of the nerve fibres, the increase of connective tissue being secondary.

The paper closes with a case of tabes dorsalis, in which there was paralysis of the legs and ataxia and paresis of the upper extremities. The autopsy showed complete degeneration of the posterior columns with the exception of a small area adjoining the posterior commissure, and also systematic degeneration of the cerebellar tracts in their whole extent and of the lateral pyramidal strands in the lumbar and lower dorsal regions. A narrow belt of marginal degeneration surrounded the cord. The affection of the lateral columns in this case was too systematic to be accounted for on the usual theory of the direct extension of the disease from the posterior columns. The only feasible explanation is that tabes dorsalis belongs to the group of primary combined systematic diseases.

W. J. Dodds, D.Sc.

Syphilis and Tabes Dorsalis.—Westphal (*Archiv f. Psych.* xi. p. 230) concludes that an ætiological relation between syphilis and tabes is unsupported either by clinical or pathologico-anatomical facts. In 75 cases, whose histories were comparatively well ascertained, he found chancres in 14, chancres and secondary symptoms in 11. Nineteen out of 20 cases occurring in women had no history of chancre, and the twentieth case was a doubtful one. In one case only were secondary symptoms present whilst the patient was under observation. Of 16 cases that came to autopsy one only showed evidences of syphilis, and in another the appearances were doubtful. Further, Westphal has never seen a case of grey degeneration of the posterior columns cured by anti-syphilitic remedies. Remak (see *Centralblatt f. med. Wissensch.* 1880, No. 43) obtained a history of syphilis in 25 per cent. of his cases of tabes, nevertheless he does not admit a direct causal connection between the two. Bernhardt admits that syphilis may

produce tabes, but it is not nearly so potent a cause as is often supposed.

Westphal reports a case in which, besides other lesions of a syphilitic nature, there was disease of the posterior columns in the upper cervical region. The medulla of the nerves had disappeared, but the axis-cylinders were intact, and in some places seemed larger than normal. The vessels were dilated and their walls thickened. There was no increase of connective tissue or proliferation of nuclei, no corpora amylacea, no cells with fatty granules. The changes were evidently due to a peculiar parenchymatous affection. A gumma was found in the posterior part of the corpus callosum. Westphal compares this case with one recorded by Schultze, in which there was a tumour of the anterior part of the corpus callosum and degeneration of Burdach's columns down to the lower dorsal region. In Burdach's columns the nerves had lost their axis-cylinders, but for the most part retained their medulla. The neuroglia was normal.

Pick on Agenesis of the Spinal Cord. (*Prager med. Wochenschr.* 1880, Nos. 15 and 16, and *Centralblatt für med. Wissensch.* 1880. No. 46.)—The results of a *post-mortem* on a child aged 14 months are reported. There was hæmorrhagic pachymeningitis, asymmetry of the cerebrum and cerebellum (the former was smaller on the right side, the latter on the left), diminution of the crus, pons, and pyramid on the right side, and discoloration of the lateral pyramidal tracts as far down as the lower dorsal regions. The discoloured tract was smaller on the right side than on the left, but this was compensated for by a small tract of discoloration in the mesial part of the left anterior column, extending to the level of the fifth cervical nerve. The discoloration was due, not to hypertrophy of the interstitial connective tissue, but to the absence in most of the fibres of the medullary sheath. The strands had been arrested at a stage in their development prior to the appearance of the medullary substance. The author remarks that if the disease (encephalitis) which causes hemiatrophia cerebralis occurs in childhood or even youth when the cord is still developing, it is quite possible that an arrest of development may ensue in the corresponding pyramidal tracts.

Under the name of atelectasis medullæ spinalis, Schiff (*Pflügers Arch.* xxi. 328, and *Centralblatt f. med. Wissensch.*, No. 39), draws attention to a similar affection in the spinal cord of dogs. He describes several varieties; atelectasis totalis, segmentalis (medulla

of nerves wanting in particular strands), marginalis (medulla wanting in fibres that form border of cord), guttata and circinata (patches and rings in which fibres have no medulla). The axis-cylinders were quite intact, and the functions of the nerves apparently unimpaired. Schiff thinks that some of the cases of sclerosis and grey degeneration of the columns in man are really instances of a congenital affection of this kind; and in point of fact, he has found in general cases of the hereditary ataxia of Friedreich, not grey degeneration of the posterior columns, but simply an atelectasis. The retention of the sensibility in such cases is accounted for by the intactness of the axis-cylinders; the ataxia is a purely cerebral symptom.

Moeli on Amyotrophic Lateral Sclerosis.—The author reports a case of this disease (*Archiv f. Psych.* Bd. x. p. 718), which presents several interesting features. (1.) The patient died of pneumonia 3½ years after the appearance of the first symptoms, during which time no bulbar symptoms showed themselves. Cases have been known in which 6 and even 14 years intervened between the spinal and the bulbar symptoms. (2.) In the lower cervical and upper dorsal regions there was sclerosis of the mesial parts of Burdach's columns, such as is found in commencing tabes. There was no history of severe pains. (3.) There was no degeneration of the anterior columns: from which it follows that this was a case of total decussation of the pyramids. Total decussation appears unusually common in these cases. (4.) There was diminution of the irritability of the muscles, but the reaction of degeneration was never obtained. (5.) There were no muscular contractures. From this we may conclude that the cornua became diseased before the pyramidal tracts.

Shaw on Subacute Myelitis of the Anterior Cornua. (*Journ. Nerv. and Ment. Disease*, Chicago, April 1880.)—A woman, aged 50, as a result of a chill, became feverish, had pains in her chest, back, and feet, and lost the use of all her limbs. A month before death her condition was as follows: Complete paralysis of the upper extremities and atrophy of the muscles of the arms and forearms, able to move her feet a little, tactile sensibility of lower extremities slightly impaired; loss of faradic reaction in the muscles; occasional involuntary passage of urine and fæces. Bulbar symptoms appeared three days before death (about three months from commencement of illness), but patient was then semi-conscious. *Post-mortem:* there was a slight meningitis in the dorsal and

lumbar regions. Throughout the cord there was degeneration of the cells of the anterior cornua, especially of the external lateral group; many of the cells were swollen and cloudy, and had apparently lost their nuclei and processes. The grey matter in the neighbourhood of the cells showed, here and there, the granular disintegration of Lockhart Clarke. In the cervical enlargement, and in it only, there was sclerosis of the columns of Goll and of the lateral columns; in the upper half of the dorsal cord there was slight degeneration of the posterior root-zones (*rubans externes* of Charcot). The author thinks the ascending degeneration of Goll was secondary to the degeneration of the root-zones.

W. J. DODDS, D.Sc.

Erlenmeyer on Nerve-Stretching in Locomotor Ataxia. (*Centralblatt für Nervenheilkunde*, No. 21, 1880.)—Langenbuch and Esmarch have described two cases of tabes treated by nerve-stretching. The idea was to relieve the patients of their acute pains; and not only did the result justify the keenest hopes entertained on that score, but, to the astonishment of all concerned, the ataxy itself was greatly reduced by the operation. Erlenmeyer discusses these cases and their various bearings, and contributes another instance of nerve-stretching in an advanced case of tabes. The patient had passed through the period of shooting-pains, and had entered upon the third, or paralytical, stage of the disease. The ataxia was excessive, the muscular sense absolutely lost, the bladder paralysed. The most energetic treatment, including anti-syphilitic courses, had failed to effect any improvement. On June 22, 1880, the right sciatic nerve was exposed by means of an incision over the sciatic notches (Erlenmeyer considers it advisable to operate as near the centres as possible). Lister's method was followed, and the wound healed by first intention. On July 3, the left sciatic was stretched. At this time the right leg had distinctly gained in strength, but the ataxy was not notably diminished. The healing of the wound on the left side gave some trouble, on account of some erysipelatous complications. On the 16th of August, the patient could stand alone against a wall, which was previously impossible, and may be considered as a proof of a gain of strength. The ataxy remains much the same; but the remnants of the shooting-pains, which atmospheric vicissitudes always aroused, have disappeared.

The comparative failure in his case is attributed by Dr. Erlenmeyer to the insufficient force exerted in stretching the nerves.

But this method of treatment in locomotor ataxy, especially at an early stage, ought to be put to the test in a systematic manner. If possible the amount of traction necessary to produce the beneficial effects should be determined objectively by means of weights, or some other means of measurement.

Müller on the Early Stage of Locomotor Ataxy (reprint from the *Mittheilungen des Vereines der Aerzte in Steiermark*, 1879).—An early recognition of ataxic tabes is of the highest importance, as it is only at the earliest stage of the disease that we can hope to do much good by our remedial measures. The ocular symptoms are frequently the earliest; and among them the author draws attention to a sudden and transitory paralysis of accommodation of one eye. This may be the only trouble present, but is often accompanied with mydriasis paralytica. These symptoms may precede the actual invasion of the disease by some years. Then dissociated paralyses of the third, of the fourth, and sixth nerves may intervene, as well as spinal myosis, which is characterised by absence of reaction to light, but not to the accommodative impulse. Or, again, mere sluggishness or absence of reaction to light may be present. Charcot has well described the progressive optic atrophy; the papilla presents the appearance of a flat depression according to Müller, and the vessels gradually become smaller. The field of vision diminishes concentrically in the two eyes unequally, the region of dimness has the shape of sectors having (not the fovea, but) the papilla as centres; and this diminution is much more readily appreciated on indirect vision. Achromatopsia may occur, first to green, then to red. In disseminated sclerosis the sense of colour is preserved. The author recommends Doucet's perimeter as the most convenient to test these defects in practice.

The lancinating pains present various peculiarities which need not be dwelt upon here; the various other defects or alterations of sensibility are also well known. Berger has shown that there may be deficient algesia to powerful stimuli, whilst weaker ones are responded to. Retardation of transmission of sensations is not very common. The sense of pressure is often deficient at an early period.

The well-known "tendon reflex" may co-exist with ataxia (in 2 cases among 82: Berger). Certain facts lead the author to suspect a period of heightened reflex preceding its abolition.

[I have now under observation a somewhat doubtful case, in which there are shooting-pains in the left leg with very diminished tendon reflex, and markedly increased reflex in the right. The right hand is the seat of progressive muscular atrophy.]

Erlenmeyer has prominently brought forward the early gastric symptoms of tabes; they either take the form of nervous dyspepsia or actual catarrh. These symptoms have the characteristical suddenness of many tabetic symptoms.

The genito-urinary organs present numerous forms of disturbance. We may note here urethral and rectal neuralgias, causing spasmodic contraction of the bladder, and tenesmus.

The well-known crises gastriques may constitute for a long time the chief feature of the disease, and may require care in their diagnosis from other stomach ailments. The pneumogastric may be affected differently, and give rise to tremendous fits of coughing. The "crises néphritiques" need only be mentioned here.

Arthropathies and osseous fragility may appear early. The pulse is usually frequent. The sweat, increased at first, becomes deficient later on, and is not readily induced by pilocarpine in the diseased regions. The ataxic symptoms are usually pathognomic of the second period. The author has noticed two cases of early peroneal paralysis.

The treatment of tabes at this initial stage consists, first, in a strict hygienic mode of life; second, in the administration of ergot and nitrate of silver, a mild course of hydrotherapy (the water to range between 20°–25° centigrade) and electricity. The latter is to be applied centrally by means of large electrodes (2 × 6 inches), using a current strength of 8–11 milliwebers; and may be also usefully applied symptomatically for relieving the ocular troubles, the gastric pains, &c. Against the latter and the shooting-pains Chapman's bags may also be used with success.

The author next discusses the anti-syphilitic plan of treatment, and condemns it—first, because it is useless; second, because he does not consider the syphilitic nature of the disease as demonstrated. We have considered this point in our review of Vulpian's work (see page 523), where the main arguments of our author are briefly reproduced. Several instructive cases bring Dr. Müller's valuable paper to a close.

Erlenmeyer on the Paradoxical Muscular Contraction. (*Centralblatt Nervenheilk.* 1880, No. 17.)—Westphal has lately (*Archiv f. Psych.* x. 1) described a phenomenon under the name of "paradoxical muscular contraction," which occurs in certain neuroses of central origin, and consists in a contraction, or rather persistent contracture, of the tibialis anticus (and other muscles) when the foot is suddenly flexed. He gave the name "para-

doxical" under the assumption that the exciting cause of the contraction was the sudden relaxation of the muscle, and therefore was the opposite of that which occurs in the "tendon reflex," where the muscle reacts to a sudden tension exerted upon it. Erlenmeyer's observations fully confirm Westphal's description of the facts; but he dissents from the explanation given by the latter. He thinks that it is not the sudden relaxation of the muscle which is the cause of its contraction, but that the phenomenon is reflex, and that it is the sudden tension of the antagonistic calf muscles which excites the tibialis anticus to contract. In proof of this view Erlenmeyer says that if, whilst the contracture of the tibialis anticus is lasting, the calf muscles are pushed down with the hand so as to relax the tension exerted upon the tendo Achillis, the contractured muscle immediately returns to its normal condition. This takes place suddenly, not gradually. The experiment may be varied in this way—that the tendo Achillis is relaxed before the ankle is passively flexed: it is then observed that the "paradoxical contracture" does not occur. It is clear, therefore, that we have not to do here with the contraction produced directly by the sudden relaxation acting as a stimulant of the muscle, but as a spinal reflex, in which the sudden extension of a muscle acts as a stimulant of its antagonist. The phenomenon is one of great interest, and likely to throw light upon the question of tendon reflex generally. The author reminds the reader of the fact, recorded by Brissaud and Richet, that in patients suffering from hysterical contractures a vigorous extension of one muscle may bring about contracture of its antagonist, due apparently to a persistent irritation of the reflex medullary centre. [Westphal has since denied the value of E.'s test.]

Schulz on Unilateral Lesion of the Cord. (*Centralblatt für Nervenheilkunde*, No. 15, 1880.)—The author relates a case in which the spinal cord had been partially divided by a stab between the transverse processes of the fifth and sixth dorsal vertebræ on the right side. Immediately after the injury the patient suffered from anæsthesia of the left leg, hyperæsthesia and paresis of the right, with loss of power over the rectal sphincter, and retention of urine. He recovered partially, and about six months afterwards presented the following symptoms: No difference in the temperature and colour of the two legs. The muscular sense is diminished in the right along with the motility. There is no ataxy nor contracture, but the movements of the right leg lack power and precision.

There is marked hyperæsthesia on the right side from the seventh vertebra and rib downwards; above this a zone of anæsthesia between the sixth and seventh ribs. The sense of touch, and especially of tickling and of pain, is increased on the right, as well as the electrical excitability of the skin. On the left the sense of touch is preserved, but that of pain has disappeared. Tendon reflex much increased on the right. Electrical reactions equal on both sides. The functions of the rectum and bladder have resumed their normal course, and there is no disturbance in the sexual power.

In Gowers' case (*Lancet*, 1877, ii.) there was loss of tactile, as well as of algesic sense. In the present instance it is probable that the former was preserved, owing to the posterior tracts not being involved by the injury. Brown-Séquard's assumption that the paths for the muscular sense do not decussate in the cord is in accordance with the facts here observed. The author thinks that the phenomena now studied of transfer may throw some light upon the hitherto obscure symptom of hyperæsthesia observed in lateral lesions of the cord.

Dujardin-Beaumetz on Æsthesiogenic Properties of Certain Woods applied to the Skin. (*Bulletin de Thérapeutique*, Aug. 1880.)—It will be remembered that the main argument for reducing the supposed efficacity of metallic applications in hemi-anæsthesia to a mere effect of "expectant attention," was that in certain cases wooden discs were found to restore sensation as well as metal plates. At the time (*cf.* 'BRAIN,' IV.) I objected to this inference, that if the effect were due to statical effects of electricity the substitution of wood did not eliminate such an influence. It has since been found that a number of other agents besides discs and the like were equally active in hemi-anæsthesia, among which vibrating tuning-forks and the like. This fact led Prof. Schiff to assume that molecular vibrations induced in the nerve might explain the extraordinary effects observed (see *Archives de Physique*, Geneva, Jan. 1880; and the criticism by Vigouroux, *Progrès Médical*, 1880, No. 36). Whatever be the value of this hypothesis, the experiments of Dujardin-Beaumetz have shown not only that wood is an "æsthesiogenic" agent, but that this property varies greatly among the different kinds of wood. They were conducted with due care; and the numerous sources of error in such observations duly guarded against. The conclusions he arrives at are that woods vary greatly in their æsthesiogenic virtues; for instance, yellow cinchona is very active, more so indeed than metals, whilst

poplar, sycamore, palissander are inactive. Between the two rank rosewood, thuya, mahogany, walnut, &c.; but the list is as yet only provisionally given.

Berger on the Nosology of Tabes Dorsalis (*Centralblatt für Nervenheilkunde*, 1880, No. 5.)—Professor B. records the results of his observations on the behaviour of *cutaneous* sensibility in some cases of locomotor ataxia. This he found to reappear after its complete loss during the progress of the disease, and whilst the other symptoms were following the usual evolution. The restoration was remarkably complete (hyperalgesia even supervening), but was confined to the sensibility of the skin, and did not extend to the deeper parts. In one case a post-mortem examination revealed a complete posterior sclerosis; and the question naturally arises as to the path followed by the cutaneous impressions in the cord.

In one case of undoubted locomotor ataxia of 10 years' standing, (in which the characteristic lightning pains, the diplopia, bladder disturbance, sensory troubles, atactic gait, were present, along with the complete loss of tendon reflex), the latter symptom was investigated most carefully, so that there cannot be any doubt about the accuracy of the statement. After a few months, during which the patient took nitrate of silver and followed a hydrotherapic course, this general condition was found markedly improved. The symptoms taken singly had much abated; and the most remarkable feature about the case was a return of the patellar reflex, especially on the left side, where even a gentle tap would excite a most lively contraction of the triceps. The author has seen such a return of the tendon reflex in cases of diphtheritic ataxy, but does not think it has yet been noted in progressive ataxy, where it proves a possibility of at least a partial restoration of the cord.

Ballet on Facial Monoplegia (*Progrès Médical*, Sept. 1880).—A Salpêtrière patient, female, age 71, sank suddenly to the ground, but did not lose consciousness. She was found to have lost all motor power over the left side of the face, without any corresponding paralysis of the arm and leg. During the day conjugate deviation to the right of the head and eyes set in; there was slight paresis of the left arm. Coma and death supervened.—Autopsy; right hemisphere: on inspection of the surface a cortical hemorrhage is seen, the size of a walnut, forming a clot of 5 grammes. It occupies the lower end of the

ascending frontal convolution, reaching below to the sylvian fissure. Methodical slicing shows that the inferior frontal fasciculus is destroyed, and the corresponding parietal fasciculus touched; but the central grey nuclei are intact.

A. DE WATTEVILLE.

Nitrite of Amyl in Epilepsy.—Dr. Maragliano (*Note di Clinica Medica*, Genoa, 1880) determined to try whether nitrite of amyl would so modify the cerebral circulation as to have a permanent effect in diminishing the fits of epilepsy. In inhaling the medicine, he used a bag of caoutchouc containing some cotton wool, which was applied to the nostrils while the patient was allowed to inspire air through the mouth. The dose given was much higher than that used by other physicians. Beginning at 10 drops he gradually mounted to 40, and he continued the inhalation as long as fifteen minutes without any instances in which harm was observed. Where the epileptic attacks were numerous, the inhalations were repeated once every hour. Dr. Maragliano quotes the experiments of Schüler, who found that in rabbits in which an opening had been made in the skull, from five to three inhalations caused a dilatation of the venous and arterial vessels of the pia mater. The smaller arteries began to pulsate, and some of them to assume serpentine curves. The brain expanded and bulged out of the trepanned hole. He notices the denial of Vulpian that nitrite of amyl has any congestive action on the brain, but finds from his own observation that the temperature of the head is increased by the action of this drug. In like manner he rejects the negative observations of Loemisch, Stammeshaus, and Pick for those of Aldridge, Tebaldi, and Gradenigo, who hold that the vessels of the retina are dilated after the use of inhalations of amyl. Ladendorf found that the temperature of the mouth was raised by half a degree C. during an inhalation. That this drug impedes the oxidation of the blood is universally admitted. He found that out of thirty cases after inhalation of amyl sugar appeared in the urine in twenty-four, and was totally absent in six. All traces of the sugar passed away entirely after three to six days. This appearance of sugar is supposed to be owing to the dilatation of the hepatic vessels. He admits that there are cases in which amyl is of no use in epilepsy, it promises most in those patients in whom the face becomes pale. Dr. Maragliano considers that the power this drug has in dilating the vessels of the brain (*la sua azione iperemizzante sul cervello*) is the cause of its therapeutic value in epilepsy.

Dr. Maragliano has made some very careful observations to ascertain the direct effect of the inspired nitrite of amyl upon the blood. He found that neither the size of the globules nor their number was affected by the drug.

The author sums up as follows :—

1. That the inhalation of nitrite of amyl methodically and persistently, and not with the view of cutting short impending attacks, causes a notable diminution of the violence and number of the epileptic seizures. This is proved by the three observations described, as well as several others in which the results were identical. The method employed by us is different from what is habitually followed, which was to cause the amyl to be inhaled after the beginning of the attack or after the aura came on in order to avert the fits. Hammond has tried the drug in much the same manner as ourselves without being able to report any benefit; but we using larger doses have obtained favourable results from the use of amyl. Some other observers have also reaped benefit, among them is Crichton-Browne, who however did not use the drug in the same fashion. He succeeded in eight cases out of ten in cutting short the status epilepticus in which fits succeeded one another without intermission, the patients remaining unconscious during the intervals.

2. That the dose of the nitrite of amyl may be raised without inconvenience to forty drops—the inhalations being prolonged for forty minutes and repeated from four to six times a day.

3. That the cerebral temperature is gradually raised from ·3 to ·8 of a degree C. during inhalation of the nitrites, on the cessation of which it again descends to the usual grade.

4. That the inhalation of nitrite of amyl in most instances causes the appearance of sugar in the urine amounting to from 1 to 2½ grammes in the twenty-four hours.

W. W. Ireland.

END OF VOL. III.

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